American Journal of Diseases of Children

VOL. 3 MARCH, 1912 NO. 3

THE USE OF A SIMPLE DUODENAL CATHETER IN THE DIAGNOSIS AND TREATMENT OF CERTAIN CASES OF VOMITING IN INFANTS*

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Some months ago I described in the pages of this journal[1] a duodenal tube for infants. This tube was introduced by mouth into the stomach, and then passed through the pylorus into the small intestine. This result was readily accomplished, the tube taking about one-half hour to pass the pylorus. The radiographs which accompanied this article showed conclusively that the end of the tube lay in the duodenum, and was not merely curled on itself within the stomach cavity. This duodenal tube, in brief, consisted of soft rubber tubing with a silver-plated, perforated leaden ball at the end, and was a modification of the Gross duodenal tube used for adults. By means of this simple device, I was able, after some practice, to gain access to the duodenum in infants in almost every instance, and gradually acquired increased facility in its use. This was, I believe, the first conclusive proof that the intestine could be reached by this route in infancy. The principle involved in this manipulation was that of gravity, carrying the leaden ball to the pylorus, aided by the onward movement of peristalsis, which swept the ball through the pyloric ring, and thence into the intestine. Although this tube served its purpose, I have laid it aside for one which is simpler, as well as more serviceable, and which has afforded me a ready means of studying the functions of the stomach and intestines from a new point of view.

In the course of my experiments it occurred to me to try whether I could obtain the same result by making use of the ordinary soft rubber Nélaton catheter, which is used so frequently in infants and in adults for the purposes of lavage and feeding. To my gratification and surprise I found that this simple instrument can be passed readily from the stomach

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*Read at the meeting of the New York Academy of Medicine, Pediatric Section, Dec. 14, 1911.

1. Hess, A. F.: A Duodenal Tube for Infants, AM. JOUR. DIS. CHILD., May, 1911, p. 360.

into the duodenum. It is indeed interesting and instructive to reflect on the fact that this catheter, which was introduced into the realm of pediatrics by Epstein in 1880 for the purpose of gastric feeding and

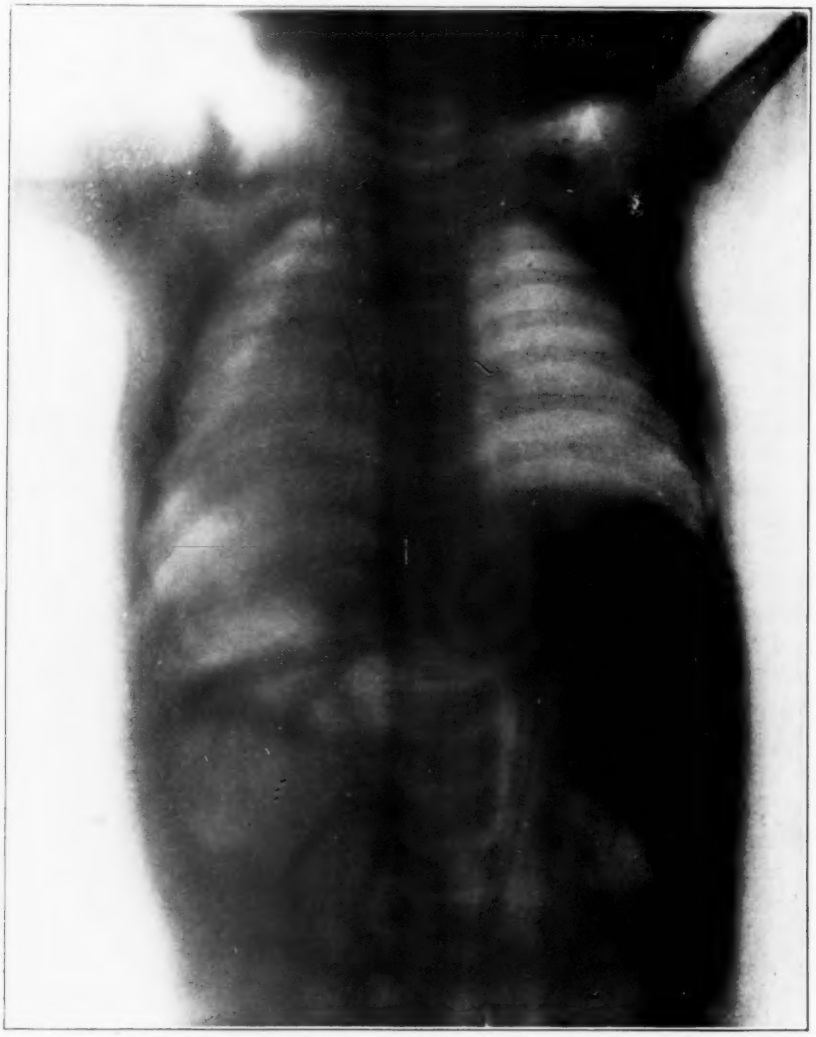

Fig. 1.—View from posterior showing catheter well within the duodenum. Note the sharp turn to the left, to the fundus, which the catheter invariably takes on entering the stomach.

investigation, has, in these many years, never been seriously thought of in connection with the diagnosis and treatment of pyloric conditions and of intestinal disturbances, subjects which have been considered of primary importance during this long period.

During the past few months I have made use of this catheter, which may in this connection be termed an "infant duodenal catheter," for these purposes on more than 200 infants of various ages, suffering from a great variety of disorders, and can at this time speak with confidence of its practicability in this connection.

In order to demonstrate beyond all doubt that the catheter gains

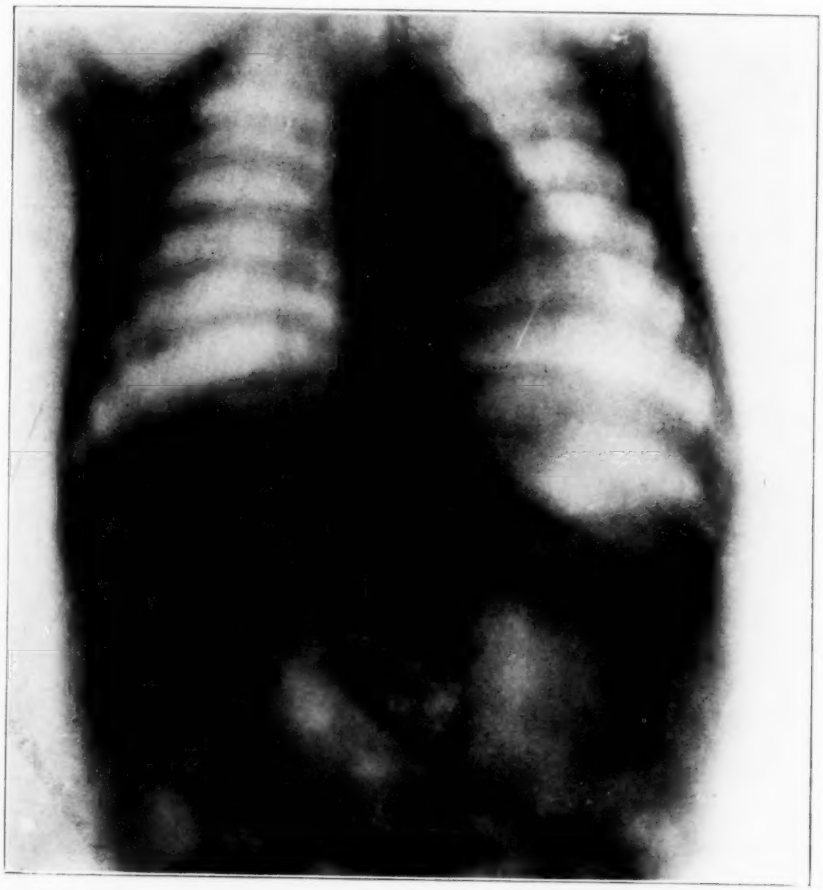

Fig. 2.—View from the anterior of a duodenal catheter deep in the duodenum.

access to the duodenum, I have once more resorted to radiographic pictures (Figs. 1 to 4), criteria which are least open to doubt, and which have enabled me to prove this point conclusively. From these pictures, for which I take pleasure in thanking Dr. Hirsch, it will be seen that the tube is well within the intestine: in fact, in one instance, seems to have passed even beyond the confines of the duodenum. It will be noted also that the catheter does not take a direct course from the cardiac end

of the stomach to the pylorus, but that just as in the case of the duodenal tube, which I previously described, the catheter on entering the stomach cavity makes a sharp bend to the left to the fundus, and then turns on

Fig. 3.—View from posterior. Duodenal feeding of 3-months-old infant; stomach empty; bismuth-milk mixture in coils of intestine.

itself to the right to reach the pyloric opening. This has been the course followed in all radiographs which I have had taken, either of this catheter or of its predecessor, the duodenal tube. This novel and unexpected picture controverts the assumption which I first considered, to explain the

ease with which I was able in infants to reach the pylorus. It shows that this success cannot be attributed to the vertical position of the stomach in infancy, nor to the fact that the tube or catheter follows an

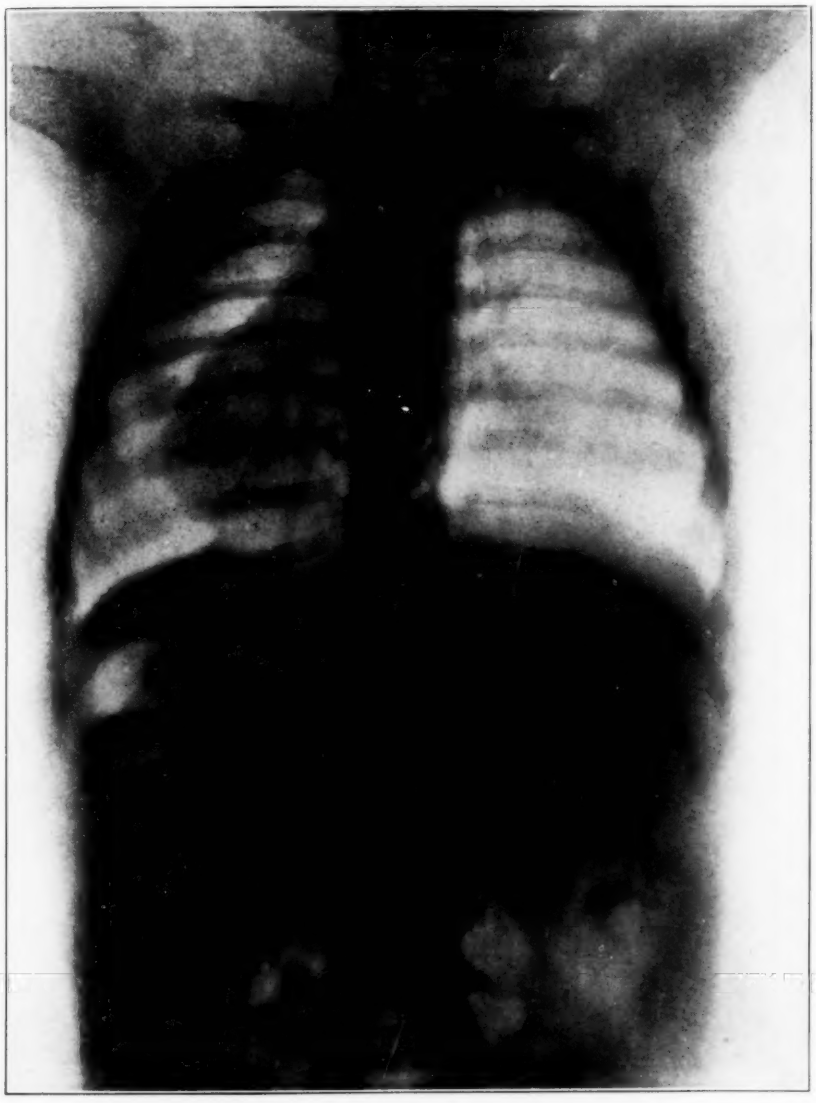

Fig. 4.—Duodenal feeding. M. C., 9 months old. Catheter and bismuth in the duodenum; stomach empty. View from the posterior.

almost vertical course in its route to the small intestine, and has led me to attribute the success of the procedure rather to the diminutive size of the stomach cavity, and also perhaps to a relative lack of tonicity of

the pyloric ring. This explanation gains added weight from the fact that a catheter of this kind cannot be used for this purpose in the adult, in whom the stomach is large, and the tube wanders far from the pyloric opening.

TECHNIC

It is difficult to describe the method of use of this simple instrument, for like all other instruments, even of a simple nature, its use can be learned only with some practice, and the descriptions are far more complicated than the procedure would warrant. For the purpose an ordinary No. 15 (F.) soft rubber catheter should be employed, preferably one with a large eye; if the baby is very small, under a month of age, a No. 14 (F.) catheter should be made use of. Before beginning we should designate points 20, 25 and 30 cm. distant from the eye. This can be done by means of pen and ink. These markings allow us to take our bearings while introducing the catheter, and as long as it is in place. The catheter is now ready for use. The only other instrument necessary is an aspirator; for this purpose either the same aspirating bulb which I described in my previous article may be used, or an ordinary aspirating syringe. The instrument is now complete. The physician, therefore, generally has an infant duodenal catheter at hand, and needs but to acquaint himself with its use.[2]

The catheter is passed in the same way as the ordinary stomach tube, that is to say, it is lubricated with water, introduced into the mouth, and without the aid of the finger is rapidly pushed down the pharynx and esophagus into the stomach. It has seemed immaterial to me whether the baby is held in the sitting position, or lies on its back, or on the side. I have generally had it placed on its back with the arms bound snugly to its side by means of a towel. In a baby 2 months old, for example, when the 20 cm. mark is about 3 or 4 cm. from the gums, the contents of the stomach begin to be evacuated. This food is generally acid, and may contain free hydrochloric acid, as shown by its reaction to congo paper, if the feeding has been given some hours previously. After the stomach is empty, if we insert the catheter gently a little further, we

2. Although a serviceable instrument can be prepared in this way by anybody wishing to make use of one, and a great deal of my work was carried out by means of a catheter which I happened to have on hand, nevertheless a catheter with two eyes at the tip, of exceptional length, and carefully marked, has additional advantages. The use of the bulb instead of an ordinary aspirating syringe has decided advantages, the greatest is that in aspirating through the bulb by means of the mouth one can in this way appreciate the amount of resistance offered by the stomach, or intestine, and thus gain valuable information as to the whereabouts of the end of the catheter. I should by all means urge anyone who intends to use this method frequently to make use of an aspirating bulb. The bulbs and the infant duodenal catheters may be obtained of Tiemann & Co., New York.

shall find no resistance until the 20 cm. mark is about at the gums, when a sense of obstruction may be felt, and the infant may gag, due to the contact of the end of the tube with the pylorus. While there is gagging or crying, accompanied by forced expiration, the tube should never be advanced, for at this time the pyloric valve, and indeed the cardia as well, is constricted and prevents further passage. If we push on a little until we reach the 25 cm. mark, we shall in most cases by this simple means have gained access to the duodenum. Indeed I have no doubt, and a recent perusal of the literature has confirmed me in this opinion, that many have reached the intestine when they wished merely to enter the stomach, and I believe that numerous reports which are supposed to record the capacity of the stomach, or to describe the nature of its contents, in reality combine data of the contents of this viscus and that of the upper part of the small intestine as well. When we consider also that pressure of the tip of the catheter on the pylorus at times causes the pyloric valve to allow duodenal contents to pass into the stomach, there is further reason for believing that errors have crept in.

Naturally, the first question is "How do we know that we have reached the intestine, and why may not the catheter be curled on itself within the stomach?" Indeed it is far easier to enter the duodenum than to learn the phenomena by which we may judge whether or not we are in the intestine; however, the more experience we gain the easier it becomes to judge of this point, and for some time I have felt very little uncertainty in this regard. There are several ways of ascertaining this fact. In the first place we must have an approximate knowledge of the distance of the pylorus from the gums or teeth. For example, this measurement for clinical purposes is about 20 cm. at one month, gradually increasing to about 25 cm. at a year.

Let us suppose that we are passing the duodenal catheter on a baby 3 months of age, and that we have gradually inserted it to the 25 or 30 cm. mark. We then halt, and watch to see whether the tube has a tendency to remain at this level or not. If you push a tube into the stomach, and have it coil up within its cavity, it will almost always, sometimes quickly, sometimes more slowly, the time depending on the fact of whether the child is quiet, or gags and resists, work its way out of the mouth, so that the 20 cm. mark comes into view again. If, however, the tube has passed into the duodenum and is held by the pyloric sphincter, it will project almost vertically from the mouth and have a tendency, unless there is marked gagging, to hold its position, and not to work its way out again. This is an important, although not pathognomonic sign of having reached the intestine. Frequently we are aided in obtaining our bearings by feeling the grip of the pylorus, an intermittent closure, when we pull slightly on the catheter.

It might be thought that one of the most useful tests for orientation would consist in obtaining alkaline fluid or bile on aspiration, and most certainly if we obtain such fluids the evidence of success is convincing, and the gratification proportionately great. However, unless we are prepared to spend one-half hour, or an hour, or at times a still longer period, we may be unable to demonstrate our success in this manner. Nor is this necessary. The duodenal contents are frequently acid, and may continue to be so, due to the fact that gastric juice is being poured out into the stomach and thence into the intestine. This may render it impossible to obtain alkaline juice, although the fluid may be golden yellow due to bile, and contain the various pancreatic ferments. Moreover we may obtain a neutral or alkaline juice without any bile, as the bile is excreted intermittently, and often at long intervals, so that if we were to depend entirely on the reaction of the fluid or on its bile content, in judging of our success in reaching the intestine, we would often consider ourselves unsuccessful, whereas in reality we had reached the desired goal.

A test which is of value, especially in the beginning of the use of the catheter, is what I have called for the sake of brevity the "retraction test." By this I mean that when we are in doubt as to whether the catheter is in the stomach or duodenum, we retract it very slowly, having an assistant aspirate through the bulb as we do so, until we have reentered the stomach cavity. In this way, if the catheter has been withdrawn from the duodenum, one of two signs may be noted: either the assistant may note a marked change of resistance to aspiration, as the end of the catheter leaves the small constricted intestinal cavity and enters the stomach (and indeed frequently the assistant may give the first signal that the tube has left the intestine), or it may be noted by the sudden change in the nature of the juices obtained. Whereas, when the tube was in the duodenum a small amount of viscid fluid, neutral or somewhat acid, was being aspirated, or indeed no fluid could be obtained, when the tube is withdrawn to, let us say, the 20 cm. mark, suddenly there is a noisy gush of fluid of an entirely different nature, perhaps containing a marked admixture of foamy saliva, generally much larger in quantity, and frequently far more acid than that which had been obtained in the duodenum. As I have said, this test tells us whether the catheter was in the duodenum or not; however, it necessitates our making a second attempt to pass the pylorus. The more we use the tube the less we have to resort to this test.

Another test, but one of much less value, is what I have termed the "papillary bile-test." By this I mean that sometimes when we withdraw the tube from the intestine toward the stomach, and reach the level of the papilla of Vater, there is a sudden gush of bile-stained fluid. This, however, is not usually the case, and the test cannot be relied on, but

should be remembered, and made use of, in case this sign is noted in the course of the "retraction test."

Like most manipulations the passage of the duodenal catheter requires some practice and cannot be learned by description, but only by personal experience. I have found that after a few attempts the technic was fairly well acquired by hospital interns. The simplest method of acquiring the technic is to pass the catheter in the way described, and control the result by means of the fluoroscope (the catheter casts a deep shadow without the aid of bismuth). However, the *x*-ray is by no means necessary, and I did not make use of it in gaining my experience. Practice — far less practice than is required to master the technic of an otologic examination — is all that is required. Indeed in very young infants it is difficult not to enter the duodenum if we do not push the catheter forward with too great haste. The younger the infant the easier is this accomplished. I have passed the catheter without difficulty on infants ranging in age from a few hours to one and one-half years. However, in a broad sense it may be stated that the younger the infant the easier it is to transgress the pyloric barrier.

In this paper in addition to describing a simple method of reaching the intestine in infants, a technic which seems to me to offer additional possibilities for diagnosis, for therapeutics and for physiologic experiment, I wish to describe how this procedure has been of value in the diagnosis and treatment of some pathologic conditions which I have encountered during the past few months.

THE CATHETER IN DIAGNOSIS

Let us consider the subject of diagnosis. There is perhaps no group of cases more puzzling at times than that characterized by persistent vomiting. From day to day our diagnosis wavers among various possibilities — chronic indigestion, pyloric spasm, beginning pyloric stenosis and other less common and even less well defined conditions thrust themselves forward in turn for consideration. For although the diagnosis in the classical case is simple, in many others we grope for weeks, and even when all is over we do not feel confident that we have solved the nature of the disturbance. In cases of this description the duodenal catheter, and at times the soft rubber ball-tube as well, has been of assistance. To cite an instance:

Case 1.—A. W., aged 2 months, weight 5¾ pounds, a premature baby, with a history of vomiting for some weeks, was admitted to Dr. Caillé's service at the Post Graduate Hospital, May 10, 1911. After admission the infant regurgitated almost all feedings; temperature was subnormal, stools yellow. Food consisted of a milk-mixture containing approximately 1 per cent fat, 6 per cent. sugar, 1 per cent. protein, of which 3 ounces were given every three hours.

May 13, 4 p. m. Catheter passed and stomach emptied of about ½ ounce of watery fluid. Some difficulty in passing tube into stomach. Duodenum could

not be entered. The duodenal ball-tube (6 mm. ball) was also tried without success. Both were held at the 20 cm. mark.

At 4:25 p. m. an attempt was made to allow the tube to find its way through the pylorus by the slow method, by peristalsis. The cardia, and more especially the pylorus, seemed irritable, the baby crying when the tube was pushed beyond 20 cm. mark. Five c.c. of markedly acid juice was obtained. At 5:30 water was put into the stomach to aid instrumentation, but of no avail; held always at 20 cm. mark. At 5:35 the tube was removed.

Epicrisis: A case of spasm or stenosis of the pylorus, combined with spasm of the cardia. Nothing was known of the history of vomiting of this case when the catheter was passed, as the infant was selected for the purpose of obtaining duodenal juice for chemical examination. It was only after our unexpected failure to pass the pylorus that we inquired the anamnesis, and learned to our great interest of the persistent vomiting. We then put 4 ounces of fluid into the stomach by lavage, but no marked gastric peristalsis was noted; 1 ounce of this fluid was forcibly ejected.

May 16, Test 2: Baby still regurgitates; food the same; fed two hours ago; temperature subnormal; stool yellow.

3:30 p. m., 6 mm. ball-tube passed; obstruction at 15 cm., which was overcome; marked acidity of food in stomach; ball again held at about 21 cm. mark, even though water was put into stomach (accessory method).

4:05, tube withdrawn, some obstruction at cardia.

4:10, 16 (F.) catheter passed, slight obstruction at 15 cm.; marked obstruction at pylorus.

4:35, marked gastric secretion; ball-tube again tried, after dipping it into cod-liver oil and pouring a small amount of oil into stomach. Failure.

Epicrisis: Again failure to enter duodenum, obstruction at cardia and at pylorus as previously noted.

May 18, Test 3: Child has vomited less since last passage of tube. Is getting mixture of milk containing approximately 2 per cent. fat, 6 per cent. sugar, and 1 per cent. proteids.

4:15, No. 12 (F.) catheter introduced into stomach, and a small quantity of very acid Congo + fluid evacuated. Obstruction at cardia at 15 cm. noted as previously.

4:45, Tube reintroduced, and at once golden, faintly alkaline fluid was obtained; 8 c.c. aspirated with the tube introduced 22 to 27 cm. (This fluid was found to contain the pancreatic ferments, amylase, protease, and lipase in considerable amounts.)

5:10, Tube removed. Pylorus probably at 21 cm. level.

5:30, 6 mm. ball-tube introduced. This could not be advanced beyond the cardia without having the baby swallow water. In 10 minutes it did not pass the pylorus.

Epicrisis: At the third test we were able to enter the duodenum, as proved by aspirating bile, and a large quantity of fluid containing all the pancreatic ferments. The pathologic condition in this case, therefore, would seem to have been a spasm of the pylorus and cardia, rather than an organic stenosis. If a stenosis did exist, it could not have been present to a marked degree.

May 27, Test 4: Baby still vomiting repeatedly. Six mm. ball-tube passed through the cardia and the pylorus by means of having the baby swallow water (accessory method). Bile obtained, and a large amount (6 c.c.) of pancreatic juice containing the three ferments. In duodenum thirty minutes.

June 1, Test 5: Baby still has propulsive vomiting. Six mm. ball-tube passed with difficulty (accessory method); a marked grip of the pylorus noted; bile obtained; tube then withdrawn.

June 9, Test 6: Infant had been given paregoric to stop the vomiting, but without effect. Condition very poor. Weight 4 pounds, 12 ounces.

4:30, 6 mm. tube passed through pylorus, by the aid of water, as proved by the "retraction test."

4:55, reintroduced. Two ounces of milk-mixture fed slowly into duodenum. June 10, Test 7: 10 a. m., inserted tube into duodenum again; 11 a. m., 8 c.c. of bile-colored fluid obtained in one hour. This fluid was alkaline, and contained the three pancreatic ferments. Pyloric tugging marked when tube was withdrawn from duodenum.

In all, seven tests were carried out on this infant. In the first two attempts we were unable to pass the catheter into the intestine. In the others this was accomplished with difficulty, and only by the aid of introducing water into the stomach. This technical resort is often very helpful, as the pylorus relaxes to allow the water to pass through, and in this way the catheter also obtains access to the duodenum. For the sake of brevity, this device, for which we do not claim originality, has been referred to in the notes as the "accessory method." In this case where a spasm of the cardia as well as of the pylorus existed, this method proved especially helpful in relaxing both sphincters. It is of interest to note that in addition to the increased amount of gastric secretion which has so frequently been observed in conjunction with pyloric spasm, there was in this case, as evidenced in the second and third test, an increased flow of pancreatic secretion as compared with the amount usually obtained. At the time this case presented itself for examination we had just begun to use the duodenal catheter, and were still employing the ball-tube. It is very probable that had we used the catheter in the examinations we should have been able to enter the duodenum more readily.

We were not in a position to attempt systematic duodenal feeding of this infant; however, the one attempt made at the time of the sixth test showed that this was feasible, and it is to be regretted that the baby could not have been given alimentation continuously by this route.

It is not our purpose to give in detail an account of all the cases of pylorospasm which we have tested by means of the duodenal catheter, nor, indeed, to discuss in this place the nature and peculiarities of this interesting conditon. We have passed the catheter on numerous infants, who on account of persistent vomiting were suspected to be suffering from this condition. In many of these cases the catheter was readily passed to and fro between the intestine and the stomach, so that the diagnosis of spasm could readily be set aside. On others a definite obstruction was encountered. One of these has specific points of interest, and, therefore, may well be recounted in detail:

CASE 2.—M. S., 3 months of age, with marasmus; never nursed; had vomited a great deal between June 27 and July 20. Gavage and paregoric used. Not vomiting at present. Weight 4 pounds, 5 ounces. Fed every two hours, 3 ounces of a mixture containing one-half boiled milk, one-half water, and 5 per cent. sugar; had been fed two hours previous to test, which was carried out to obtain pancreatic ferments.

August 18, Test 1: 2:20, catheter introduced into stomach, which was found to be empty. 2:25, No. 14 (F.) catheter passed readily into duodenum. Some warm water was given. In the course of thirty minutes about 13 c.c. of alkaline, greenish, slightly viscid fluid was obtained (subsequent examination showed this to contain lipase and protease to a marked degree; the amylase test was lost). 12:55, tube removed. Clear acid fluid obtained in stomach.

Epicrisis: A normal case except for the excessive secretion of duodenal fluid.

September 12, Test 2: Weight 4 pounds, 8 ounces. Appears nervous and cries a great deal. Since September 3 infant has been vomiting, and for the past three days has vomited all food. It is given 4 ounces of skimmed milk every two hours. For the past twenty-four hours the infant received 4 minims of paregoric every four hours, as from July 12 to July 17; was fed about two hours previous to test. 2:20, No. 14 (F.) catheter passed. Stomach empty. 2:35, catheter again passed into stomach. Same obstruction at cardia, which was passed; the sphincter was felt to grip the catheter when withdrawal was attempted. Could not pass the pylorus even with the aid of the "accessory method." Marked flow of gastric juice, giving a strong reaction for free hydrochloric acid (Congo).

3:40, Catheter removed. Grip of cardia again clearly felt on withdrawal.

Epicrisis: Spasm of cardia and pylorus with marked gastric secretion.

September 15, Test 3: Weight 4 pounds, 15 ounces. Regurgitates much less. Has gained 7 ounces in four days. Still obtains paregoric in same doses. No 16 (F.) catheter at once passed into duodenum and bile obtained. Child much quieter.

Here we have the record of a child which gave a history of vomiting during the second month of life, but when examined in the third month seemed normal, as far as our test is concerned, except for an excessive duodenal secretion. About two weeks later it began again to regurgitate persistently, and when tested for a second time gave unmistakable signs of cardiospasm and pylorospasm. The spastic condition developed under our observation. A third passage of the catheter, carried out a few days later, revealed the fact that the spasms had relaxed, and coincident with this change in physical condition, a decided decrease in the regurgitation was noted, as well as a general improvement and gain in weight. This improvement took place subsequent to the passage of the catheter. However, in spite of the fact that it retained its food it gradually sank, and died of marasmus about two weeks later. Duodenal feeding was not attempted, as there was no one at hand to carry it out satisfactorily.

It will be seen from a survey of these two cases, and I may add that other instances corroborate this view, that the duodenal catheter enables us to make an absolute diagnosis of pyloric spasm, and to sharply differentiate this condition from vomiting not spastic in nature. A single test generally has been sufficient to draw this distinction. However, if we merely fail to enter the duodenum in a case, we would certainly err to attribute this failure to spasm. But if we are sure of our technic, and have passed the catheter in numerous instances without difficulty, and then meet with a case offering persistent obstruction at the level of the pylorus, we are justified in attributing this hindrance to pyloric spasm. This can be our conclusion only if we finally overcome the obstruction.

If we fail to enter the intestine, a single test must be considered insufficient, as the failure may be due to other causes, especially if our experience has not been wide. It is in such cases as I have detailed at length, in which the catheter is repulsed again and again at the same level, where we finally feel the resistance give way and the catheter pass, where there is a marked grip or tugging when the attempt to retract is made, that we may venture a diagnosis of spasm. It is not possible to differentiate between a simple spasm and a true stenosis of so mild a grade that it permits of the passage of a No. 15 (F.) catheter. However, if we are unable to enter the intestine after repeated attempts in the case of an infant showing symptoms of pyloric spasm or stenosis, the latter must be looked on as the more likely pathologic condition.

In most of our cases in addition to the spasm of the pylorus, we have encountered a spasm of the cardia. That this functional disturbance has not been met with frequently by others can be accounted for only by a difference in technic. If a catheter is forcibly pushed down the esophagus into the stomach, a moderate increase of resistance of the cardiac sphincter will readily be overcome, and just as readily escape observation. However, if the catheter is inserted slowly and gently, it will be noted that there is frequently a definite resistance persistently met with at a point which is about 15 or 16 cm. from the lips, whenever we attempt to enter the stomach. When a very soft rubber tube, such as the duodenal ball-tube, is employed, we find this obstruction intensified, so that, as in the two tests in Case 1, it becomes impossible to pass the cardia unless we relax this sphincter by having the baby swallow some liquid (accessory method). This interesting syndrome of pylorospasm adds weight to the argument of those who favor a neuropathic origin for this condition. In the case of an infant 7 weeks of age suffering from pylorospasm, it was found that frequently the food did not even enter the stomach, but was checked at the cardia and quickly regurgitated. In this case it was impossible to pass the soft tube into the stomach. When the catheter was inserted into the stomach this organ was found to be absolutely empty, a condition which in my experience is very exceptional.

Another observation which is interesting, and which has not, to my knowledge, been previously described, is an anomaly of secretion in the duodenum frequently accompanying pylorospasm. For some time it has been realized that many cases of spasm of the pylorus are accompanied by a marked increase in secretion of gastric juice. In the two cases which I have detailed, this was true. In addition there was an increase in the secretion of the duodenal fluid. In the first case in Test 3, the first one in which I was able to enter the duodenum, 8 c.c. of fluid was aspirated within twenty-five minutes; in the next test 6 c.c. was obtained in thirty minutes, and in the seventh test, the only other in which the juice was

collected, 8 c.c. of alkaline fluid was secreted in one hour. Comparative tests on other infants show unequivocally that this far exceeds the normal total of secretion for such short periods. The second case gives evidence that this hypersecretion, or "duodenal succorrhea," as it may be termed, may persist even when the spasm is not present. It will be seen that in the first test of Case 2, which, it will be remembered, was undertaken when the baby was no longer vomiting, although gavage had been necessary about three weeks previously, a marked duodenal hypersecretion was noted; 13 c.c. of alkaline, greenish juice being obtained in thirty minutes. The fluids in all these instances were examined for the three pancreatic ferments, and found to contain them rather in excess, so that we must conclude that in cases of pylorospasm accompanied by gastric hypersecretion there is often also a pancreatic hypersecretion. It is well established that the gastric juice serves to stimulate the pancreatic juice, but tempting as this explanation appears, it may be that both secretions have a common exciting cause.

I have intimated that some cases of pylorospasm are not accompanied by gastric hypersecretion. Most observers now distinguish between these two groups of cases. The case of the 7-weeks-old infant whom I referred to in connection with the obstinate cardiospasm, well illustrates this distinction. There was only one test performed, and this showed the stomach absolutely free of hydrochloric acid both at the beginning and at the end of the test. In this instance there was likewise a hypersecretion of duodenal fluid, instead of the succorrhea which we encountered in the previous cases, so that using also the pancreatic secretion as a criterion we can differentiate two groups of cases.

USE OF THE CATHETER FOR THERAPY

In infants persistent vomiting may result in extreme inanition, or even death. We see this most frequently, perhaps, in cases of pyloric stenosis or of pylorospasm. In spite of all our efforts to relieve the spasm or to provide an appropriate food the vomiting persists. But in addition to this group of cases there are others less sharply defined, termed by some merely "persistent vomiting," for want of a more exact term, in which the infant dies of inanition, and the stomach is found normal at autopsy. Gittings[3] recently reported three such cases. Again, during or shortly following the acute period of some of the infectious diseases, such as pneumonia, vomiting may supervene and threaten or end the life of the weakened infant. I believe cases of this description are more frequent than is generally realized.

The main treatment of vomiting in infants, whatever its cause, may be summarized as dietetic, the selection of an appropriate food, or as mechanical, which includes the washing of the stomach, as well as feeding

3. Gittings, J. C.: Arch. Pediat., August, 1911, p. 661.

by means of the tube directly into the stomach. But even these methods are not always successful, and many infants succumb to marasmus or finally to some secondary infection. It is in these extreme instances in which the ordinary measures fail, that I believe duodenal feeding has a place. The two radiographs (Figs. 3 and 4) which I have had reproduced clearly demonstrate that this procedure is feasible. They show us the catheter in the duodenum, and the bismuth-milk mixture in the various coils of the intestine. Figure 3 shows this particularly well. In adults Einhorn has employed duodenal feeding. For the purpose of feeding, the catheter is introduced in the usual way, and is inserted to about the 40 cm. mark, if this can be accomplished without force. When the catheter has entered this distance, I have refrained from moving it in and out, in order not to irritate the intestine. For the same reason, as well as on other physiologic grounds, I have always had the milk heated to about 100 F. It is probably also of advantage, judging from experiments *in vitro,* to have the milk peptonized, although I have many times given raw milk in this way without observing signs or symptoms of indigestion. The infant must be fed slowly. In some cases this precaution is forced on us, as the fluid enters the intestine with difficulty, and it even becomes necessary to force it onward by means of pressure, which is most readily carried out with the aid of the aspirating bulb. But where the fluid has a tendency to run rapidly through the funnel, which is attached to the catheter, the flow should be obstructed, and ten to twenty minutes should be consumed for the entire feeding. Otherwise we run the risk of inciting antiperistalsis by overdistending the gut, and of causing regurgitation of the fluid from the intestines into the stomach. Indeed this happens to an inconsiderable extent whenever we introduce large quantities of liquid directly into the intestine. We may then find that some of it, perhaps an ounce, has found its way back to the stomach, and may later be regurgitated, although the bulk of the food has been satisfactorily retained.

I have had an opportunity of feeding a considerable number of infants by this method. In most cases I have done so but once or twice, because further opportunity did not present itself. In these instances the report has been that the food was entirely or almost entirely retained. Two urgent cases in which "duodenal gavage" was continued for some days seem worthy of a detailed report:

CASE 3.—B. B. was admitted September 6 to Beth Israel Hospital, suffering from lobar pneumonia. The child was 10½ months of age, had been ill five days, and gave a history of cough, dyspnea and fever. By September 11 the temperature was normal, falling by lysis. On September 7 the infant began to vomit its feeding, which consisted of 6 ounces of milk, 2 ounces of barley water, and 4 per cent. sugar every four hours. The vomiting increased in frequency, until by September 13 the child was retaining very little nourishment. Smaller quantities of food were given, lavage followed by gavage, sodium citrate, small doses

of codein before feedings, but all to little purpose, and the child sank into a very critical condition. Accompanying a rapid loss of weight, the temperature dropped and remained subnormal (Figs. 5 and 6). On the morning of September 15, I introduced 4 ounces of milk mixture (three-quarters milk, one-quarter barley, sugar 2 per cent) into the duodenum and it was retained. The day following I repeated this procedure with favorable result, so that regular duodenal feedings every four hours were begun. This was discontinued after four days when it was found that the infant, in addition to having gained in weight, was able to retain most of its food when given directly by mouth. The child did not vomit frequently after this date, and was discharged cured November 14. The feeding was carried out as above described.

This case showed the importance of introducing the food slowly, as whenever it was allowed to stream rapidly through the catheter, marked

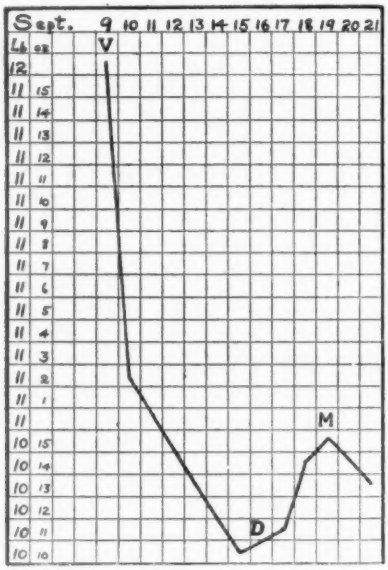

Fig. 5.—Chart showing marked loss of weight in Case 3 during period of vomiting, with gain in weight as the result of duodenal feeding. V, vomiting; D, duodenal feeding; M, feeding by mouth.

gagging would result with a regurgitation of some of the milk. In fact even if the procedure is performed with care there may be a reflux of a small quantity of milk into the stomach. For this reason it is better to limit the amount of feeding to from 1 to 4 ounces according to the size of the child, and the ease with which the milk can be successfully introduced. This is advisable also because these infants are generally suffering from a marked grade of inanition, and it is, therefore, unwise to allow them suddenly large quantities of food.

A second case in which the vomiting was more violent and the inanition more extreme is the following:

CASE 4.—L. N., 18 months old, was admitted to Beth Israel Hospital, September 13, on account of enteritis. The intestinal disturbance yielded to "Eiweissmilch," and all seemed well, when on September 27 the child began to vomit and to give signs of nephritis. By September 29 the child had lost 1¾ pounds, its tongue was dry, its expression anxious; leukocyte count 21,000. Lavage was carried out, and small doses of codein given before feedings. By the day following, it had lost 11 ounces more, that is, almost 2½ pounds in four days. Lavage had proved of very temporary value. The next day's weighing showed a loss of 2 ounces more (Fig. 7). The child's condition was very serious, its eyes were sunken, its tongue dry, acetone was present in the urine, and all food was rejected whether given by spoon or by tube. The vomiting was markedly projectile in type; when the child lay on its back the milk was ejected in a vertical column of about 3 inches. Food in small quantity, and in weak dilution was resorted to, but all was rejected, even small quantities of water.

October 1 duodenal feeding was attempted on two occasions with success, and the day following four feedings were given in this manner, and one feeding of 1 ounce by spoon. The duodenal feedings consisted of 6 ounces of equal parts of milk and water, with 3 per cent. sugar. This quantity, as I now realize, was unnecessarily large, and generally 1 to 2 ounces were rejected. The 1 ounce feeding by spoon was not retained. October 3, although the child had not gained, it looked better; by the next day it was found to have gained 2 ounces; and by

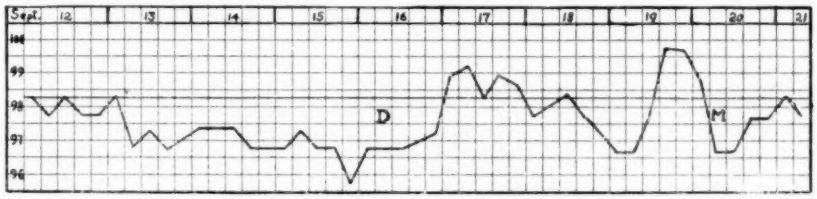

Fig. 6.—Chart showing subnormal temperature in Case 3 during period of vomiting, with rise of temperature as the result of duodenal feeding. D, duodenal feeding begun; M, mouth feeding.

October 6 an additional 6 ounces. The child looked much better, the urine no longer contained acetone, the pylorus seemed relaxed, so that feeding by spoon was once more attempted. The child now retained small quantities of food, and duodenal gavage was entirely abandoned. It was discharged cured from the hospital November 16.

The nature of this sudden attack of vomiting is obscure, but this is not of great moment from the viewpoint of the question of feeding. It was certainly spastic in nature, as proved both by the typically projectile character of the vomiting, and by the resistance which the pylorus offered to the catheter. This resistance became noticeably less after the first few introductions of the tube. It is difficult to judge at present whether this was the result of a dilatation and relaxation due to the instrumentation. It is possible that the passing of the catheter reduced the spasm of the pylorus. This seemed to be true in this instance, as well as in Case 2, described above, in reference to the diagnosis of spasm, and in cases observed by others following the passage of the catheter.

I cannot but feel that the tide was turned in the life of this infant by means of duodenal feeding. There was a sudden change for the better, and although some of the food was regurgitated, as the quantity given was too large, the child gained in weight and strength. It should also be noted that we were able in a few days to abandon feeding by the catheter for regular feedings with the spoon.

It gives me pleasure to acknowledge my indebtedness to Dr. Caillé, and to Dr. Chapin of the Post Graduate Hospital, for the privilege

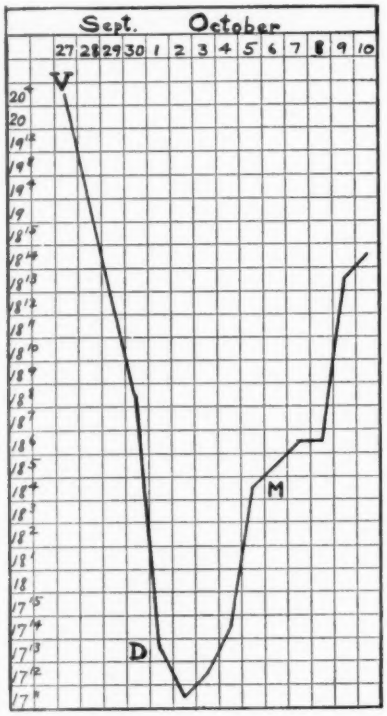

Fig. 7.—Duodenal feeding. Showing loss of weight in Case 4, due to vomiting, and gain following feeding through duodenal catheter. V, period of vomiting; D, beginning of duodenal feeding; M, beginning of mouth feeding.

accorded me of carrying out a large part of this work in their wards, and of thanking Miss Gertrude Fisher for her able assistance.

SUMMARY

In infants it is possible without difficulty to insert a soft rubber Nélaton catheter (No. 15 F.), past the pyloric sphincter and into the duodenum. The catheter is introduced in the same way as the ordinary stomach-tube, and, after some experience is acquired, the technic employed becomes almost as simple.

In principle the catheter differs from the duodenal tube previously described by the author mainly in that this instrument does not depend on gravity or peristalsis to direct it to the pylorus. The mere force of inserting it propels it along the natural path of the food to the pyloric opening. This fact not only enables it to be introduced readily and surely, but gives to it the additional advantage of a probe, a pyloric probe with which we may test the tonicity and irritability of this sphincter. Radiographs show that the catheter invariably, on entering the stomach, bends sharply to the left to reach the fundus, and that therefore the more vertical position of the stomach of the infant, does not account for the ease with which the duodenum is entered.

It is probable that unknowingly others have entered the intestine by this method, and that in many instances reports as to the contents or the capacity of the stomach in infancy have been subjected to this source of error.

By means of the catheter we can readily diagnose pylorospasm, and differentiate it from vomiting due to other causes. In the case of spasm we meet with a persistent resistance encountered at the same point whenever we attempt to advance the catheter; this is frequently accompanied by irritability of the pylorus. The spasm may be felt to relax suddenly and enable us to enter the intestine.

Marked pyloric stenosis can be diagnosed from the failure to transgress the pylorus after repeated attempts. A mild degree of stenosis, so slight as to allow of the passage of the catheter, cannot be differentiated from simple spasm.

Cardiospasm frequently accompanies pylorospasm. This sign has been frequently overlooked, due to the too forcible insertion of the stomach tube. If a soft rubber tube, such as was originally used, is introduced into the esophagus, it may be found even impossible to enter the stomach. Frequently as the result of this spasm the food does not enter the stomach, being checked at the cardia.

Just as a marked gastric secretion frequently is associated with pylorospasm, so also is an increased duodenal secretion (duodenal succorrhea). This secretion is found to contain protease, lipase and amylase to a marked degree, so that in this connection we may speak of a pancreatic hypersecretion or succorrhea.

There are cases of cardiospasm and pylorospasm unaccompanied by increased gastric secretion. In a case of this kind there was likewise no pancreatic hypersecretion.

The catheter is of value in the therapy of pylorospasm. Its passage through the pylorus seems to relax the ring and in this way to diminish the vomiting. It would seem of advantage to test this method of dilating the pylorus, and to pass the catheter frequently in such cases.

Another form of therapy consists of duodenal feeding. Radiographs show that this is feasible. It should be reserved for such cases as do not retain food given by gavage, and the food should be given slowly and in not too large amounts. In cases of this kind it has been found of great value.

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A STUDY OF THE CEREBROSPINAL FLUID AND BLOOD IN ACUTE POLIOMYELITIS *

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THE CEREBROSPINAL FLUID

There are two ways in which a careful study of the cerebrospinal fluid in acute poliomyelitis may help to throw light on the disease. On the one hand, the changes observed may facilitate early diagnosis and thus enhance the value of any method of treatment which may be discovered in the future, and on the other hand, the variations in the character of the fluid, as observed during the progress of the disease in each case, may give some information as to the usual course of the process and thus be an aid in determining how far any given remedy is effective or to what extent the natural course of the disease may be influenced. The following study was thus made with the twofold object of determining whether the examination of the spinal fluid may be an aid in early diagnosis, and what changes occur in the fluid during the course of the disease.

Previous investigations along this line have not been very extensive. Most writers report only a few cases, and those who have apparently made a large number of lumbar punctures have not submitted the fluids to very careful examination. The results obtained are somewhat variable, but in general the fluid is said to contain an abnormal amount of albumin and a sediment consisting of lymphocytes, large mononuclear cells and occasionally polymorphonuclear cells. Important work has been done by Gay and Lucas,[1] whose contributions are the most valuable in this field. Their studies have embraced the spinal fluids from monkeys in the incubation period, the prodromal stage and the acute stage, and from eleven human cases in the preparalytic stage. Besides noting the character of the cells, the number of cells per cubic millimeter has been estimated. In the eleven early cases the cell count varied from fifty-five to 580 per cubic millimeter and the percentages of mononuclears from seventy-five to 100. Chemical examinations were omitted. Sophian[2] reports that the globulin test is positive in the early stages.

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*This paper will form part of a monograph on the clinical aspects of acute poliomyelitis to be published by the Rockefeller Institute for Medical Research.

*From the Hospital of the Rockefeller Institute for Medical Research, New York.

1. Gay and Lucas: Arch. Int. Med., 1910, vi, 330; Lucas: AM. JOUR. DIS. CHILD., 1911, i, 230.

2. Sophian: Arch. Pediat., March, 1911.

Inasmuch as previous work by Wollstein,[3] Römer and Joseph,[4] and Gay and Lucas[1] failed to demonstrate the presence of any specific antibody in the spinal fluid in acute poliomyelitis, we have confined our study to simple chemical and cytological examinations.

It has been our object to see the cases as early as possible after the onset of the disease, to make a lumbar puncture on admission to the hospital, to repeat the puncture every two to four days in the early stages, and in the later stages to make a puncture every five to ten days while the case was under observation, or until the fluid became normal. We have examined in all 233 fluids from sixty-nine cases. The number of cases seen in the first week of the disease (dating from onset of symptoms, not of paralysis), was forty-three, in the second week forty-five, in the third week forty, and in the fourth week thirty. The number of cases seen later in the course was much smaller. It is to be regretted that the number of preparalytic and abortive cases was so small — of the former, four, and of the latter, only two were seen in the active stage. It was, however, rare that cases were referred to the hospital until a positive diagnosis, based on the appearance of paralysis, had been made by the attending physician.

The methods used for the examination of the cerebrospinal fluid have included estimation of the pressure, determination of the number of cells per cubic millimeter and the types of cells present, estimation of the chlorids and observation of the power of reducing Fehling's solution, and on the presence of globulin. Globulin was tested for by the butyric-acid method of Noguchi and the relative strength of the reaction noted. Thus "very slightly plus" (v. s. +) indicates a faint precipitate, which is, however, definitely more than that obtained with normal spinal fluids; "slightly plus" (s. +) is the term applied to reactions giving a well-marked cloud or a slightly flocculent precipitate; "plus" (+) signifies a precipitate coming down in large flocculi; and "double plus" (+ +) a heavy flocculent precipitate. The reaction was performed with accurately measured amounts of spinal fluid and of reagents, and the reading made after five to ten minutes' standing.[5] The gross appearance of the fluids was very constant. Almost all were clear, colorless and watery. A few, only, showed the slightest opalescence. On standing, a delicate web-like clot formed in a small proportion of cases. Clot formation was most frequently seen in the early stages of the disease. It bore no apparent relation to the globulin content of the fluid.

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3. Wollstein: Jour. Exper. Med., 1908, x, 476.

4. Römer and Joseph: München. med. Wchnschr., 1910, lvii, 568.

5. To 0.2 c.c. spinal fluid is added 0.5 c.c. butyric acid solution (10 per cent. butyric acid in 0.85 per cent. solution of sodium chlorid). The mixture is boiled, 0.1 c.c. normal sodium hydrate solution added, and then boiled again.

The results obtained by several of these methods may be dismissed briefly. Pressure determinations were made in a number of cases and the readings were usually above normal. The pressure was read in milimeters of spinal fluid in a tube of 1.0 mm. bore. No extremely high readings were obtained, and in some instances normal pressures were seen during the early stages of the disease. On the whole, the estimations of pressure were unsatisfactory, as the patients were usually crying children.

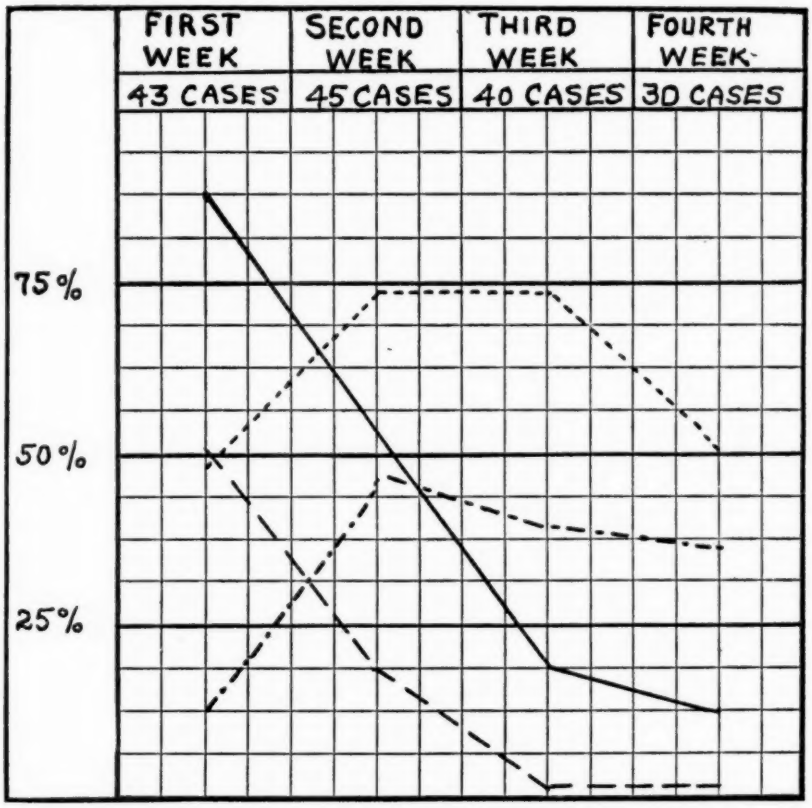

Chart showing variations in cell-count and in globulin content of cerebrospinal fluid in infantile paralysis. ——— Percentage of cases with cell-count above normal. — — — Percentage of cases with cell-count above 50 per c.mm. - - - - Percentage of cases with globulin "s+". —.—.—. Percentage of cases with globulin "+" or over.

The chlorids were estimated in a series of cases with the idea that they might throw light on the inorganic substances in the fluid. No marked deviations from the normal were observed. Every fluid examined showed a power of reducing Fehling's solution which was, roughly speaking, normal. This reaction serves as a point of differentiation from many

fluids in cases of meningitis, in which the reducing substance may be absent.

The type of cell present in the fluid was almost always the mononuclear. A large number of differential counts showed that the relative number of the different types of mononuclear cells was of comparatively little importance, so in later observations we were content with merely noting the relative number of mononuclear and polymorphonuclear cells. In several instances, numbers of very large phagocytic cells containing vacuoles and broken-down cells were seen. Once this occurred several weeks after the onset of the disease, in association with a sudden rise in the cell count resembling that seen in acute meningitis. Polymorphonuclear cells, except in small number and probably depending for the most part on a slight admixture of blood, were, except in a very few instances, absent after the first week of the disease. Early in the disease, however, and in the preparalytic period, the polymorphonuclears sometimes outnumbered the mononuclears and made up 80 to 90 per cent. of the total.

The results described by Flexner and Lewis[6] of a study of the cerebrospinal fluid of a monkey after inoculation with the virus of poliomyelitis are interesting. Twenty-four hours after inoculation the fluid contained "a considerable number of small cells, hardly exceeding a lymphocyte in size but showing a polyform nucleus, a few lymphocytes and some red corpuscles. At the expiration of forty-eight hours, the white cells have increased in numbers, but the cells with polyform nuclei still predominate. At the expiration of seventy-two hours, a large number of mononuclear cells have appeared and the fluid presents a striking opalescent appearance. On the day of paralysis the fluid tends to be only slightly cloudy and contains a mixture of large and smaller (lymphoid) mononuclear cells and a few cells with polymorphous nuclei." Lucas, too, found in the monkey during the prodromal stage "a marked increase in the cells, often reaching 1,000 per cubic millimeter. In this stage, also, polynuclears are still present, in some cases as high as 60 per cent., though the large mononuclears and lymphocytes were very evident. In the early acute stage the increase in cells is very marked. The cells are now, however, mostly of the lymphocytic or very early form of cells, and sometimes very hard to place, as they are apparently undifferentiated cells." Of his eleven preparalytic human cases, however, only one showed less than 87 per cent. mononuclears, and that had 75 per cent. mononuclears. A return of polymorphonuclears later in the disease, as was reported by Lucas as occurring in monkeys, was not noticeable in our human cases.

The cell count was made immediately after the fluid was obtained. The first portion of fluid obtained was used, but controls made by com-

6. Flexner and Lewis: Jour. Am. Med. Assn., 1910, liv, 1140.

paring counts of early and late portions of the fluid showed no important discrepancy. While the normal number of cells per cubic millimeter of spinal fluid is usually given as three to five, we have considered the upper normal limit as ten to twelve. In general it may be said that the highest cell counts are found in the early days of the disease, and that there is a progressive falling off as time advances. Thus of forty-three cases in the first week, twenty-three showed fluids with over fifty cells per cubic millimeter and thirty-eight with cell counts above normal. Two cases showed in the prodromal period 990 and 650 cells, respectively. Of forty-five cases in the second week, in eight the counts were over fifty, and in twenty-three cases they were above normal; of forty cases in the third week, in only one was the count over fifty, and only in eight above normal. On the other hand, six cases in the first week, twenty-two cases in the second week and thirty-two of the forty cases in the third week showed normal counts.

In contrast to the cell count which thus tends to be high in the early part of the disease, the globulin content is usually low in the first part of the acute stage, especially as compared with the cell count. It rises during the second and third weeks and then gradually falls, though frequently globulin is present long after all acute symptoms have passed (see chart). We have seen a strong (+) globulin reaction on the forty-sixth day of the disease. During the first week, only six of forty-three cases showed at any time a globulin reaction of + (see above) or more. During the second week twenty out of forty-five showed a + reaction, in the third week sixteen out of forty, and in the fourth week nine of thirty cases. On the other hand, sixteen cases in the first week showed an absolutely normal globulin reaction. Five cases in the second week and only three in the third week were negative for globulin. Most cases, even on discharge, gave a globulin reaction which varied from slightly to markedly above the normal. In eleven cases the fluids were followed until both cell count and globulin reaction became normal. The return to normal occurred in the third week in two cases, in the fourth in two cases, in the fifth in one case, in the sixth in two cases, in the seventh in three cases, and in the tenth week in one case.

Of the sixty-nine cases studied, only two never showed any abnormal fluids. In each of these cases the fluid was examined but once, on the twenty-fifth and thirty-third days of the disease, respectively. One case showed, in five fluids taken between the ninth and forty-first days of the disease, no evidence of abnormality other than the slightest positive globulin reaction (v. s. +), and another, in three fluids obtained on the ninth, sixteenth and twenty-third days, gave similar results. Four cases, including one abortive case, showed fluids which, except for the slightest

globulin reaction, were normal at the first tapping and subsequently became more definitely positive.

The commonest type of fluid seen in the whole series is one with normal or slightly increased cell count and a well-marked globulin reaction. There are, however, two other distinct types of fluid met with. The one is a fluid with a high cell count and a normal or very slight globulin reaction, and the other is a fluid with a normal or low cell count and a very marked globulin reaction. Twenty-six cases showed at some time a globulin of not over s. + and cell count of fifty or over. Twenty of these occurred in the first week, and in twelve the count was over 100 cells per cubic millimeter. On the other hand, sixty-two fluids from thirty-four cases gave a globulin reaction of + or more. In thirty-three of these the cell count was normal and in fifty-five it was not above forty per cubic millimeter. That the two types of fluid are fairly distinct is shown by the fact that only five cases showed fluids with both a high cell count (over fifty), and even a moderately high globulin. Each type of fluid is, moreover, quite definitely characteristic of a stage in the disease. The cellular exudate is almost always associated with the earliest days of the acute stage, the albuminous exudate with the latter part of the acute stage. It is interesting that of ten cases seen within the first three days after the onset of symptoms, all but one had a cell count of over fifty (one was thirty-seven), and six were over 100. On the other hand, six gave a negative globulin reaction. In four the reaction was s. +.

DIAGNOSIS

As regards early diagnosis, the cases examined before the onset of paralysis are of the greatest interest. Six cases were seen in what may be termed the prodromal period. Four of these developed paralysis later and two proved to be of the abortive type. In one case a single lumbar puncture was made on the fourth day after the onset. The fluid gave a cell count of 650 per cubic millimeter, 90 per cent. polynuclears, and a + globulin reaction. Three days later the child suddenly developed paralysis and in five hours died of respiratory failure. A second case, the sister of a child already in the hospital, was seen on the day after the onset of symptoms. The spinal fluid contained 990 cells per cubic millimeter, 90 per cent. of which were polymorphonuclears, and the globulin reaction was s. +. On the following day one leg was paralyzed and the cell count of the spinal fluid was 627 per cubic millimeter with 80 per cent. mononuclears. The next day both legs were paralyzed and the cell count was 1,221 per cubic millimeter, with 92 per cent. mononuclears including many large phagocytic cells. The globulin reaction was +. Thus two cases seen very early in the course of the disease showed fluids with unusually high cell counts, and with a marked pre-

dominance of polymorphonuclear cells. In one of them the change of the cell picture from the polynuclear type to the mononuclear type could be followed coincident with the onset of the paralysis. Three other cases in the prodromal period, one an abortive case and two becoming paralyzed on the day of the first lumbar puncture, gave cell counts of thirty-seven, ninety-four and 113 per cubic millimeter, with 100 per cent., 93 per cent. and 83 per cent. of mononuclear cells, respectively, and globulin reactions which fell within normal limits. Finally in one abortive case lumbar puncture on the second day after the onset of symptoms gave a wholly normal fluid. On the third day the cell count was sixty-two per cubic millimeter, with 89 per cent. mononuclears and a s. + globulin reaction. On the sixth day the cell count was fifty-nine per cubic millimeter, all mononuclears, and globulin s. +. On the eleventh day the spinal fluid was again normal. That this was in fact an abortive case of poliomyelitis was proved later by showing that the patient's blood-serum was capable of neutralizing active virus when mixed with it and injected intracranially into a monkey.

In these six cases, then, examination of the spinal fluid gave evidence which was helpful in making a diagnosis of poliomyelitis before the onset of paralysis. Similar evidence was afforded by eleven cases of Lucas and by one of Frissell.[7] In none of these twelve cases, however, was a large percentage of polynuclear cells found. But it seems of importance to call attention to the fact that a fluid whose cytology resembles that found in cerebrospinal meningitis and other types of purulent meningitis may also occur in the early stages of poliomyelitis.

The question arises as to whether there is any relation between the situation and extent of the paralysis or the severity of the disease and the character of the spinal fluid. Conclusions on this point cannot be drawn from the number of cases seen by us. It would seem, however, that in cases in which the paralysis is limited to arms or to cranial nerves, the lumbar puncture fluid is rather apt to show less deviation from the normal than where the legs are involved. This is, however, far from being a constant finding. Several cases which had acute symptoms (pain, irritability), lasting for an unusually prolonged period, gave an unusually large and persistent globulin reaction. Moreover, the disappearance of the acute symptoms was frequently coincident with a diminution in the globulin in the spinal fluid. The fatal cases showed nothing in the fluid on which to base a bad prognosis.

Since the infecting organism in poliomyelitis is too small to admit of its being seen, even if it is present in the cerebrospinal fluid, and since the more complicated biological tests have failed to prove the presence of antibody in the fluid, the diagnostic value of examinations of the

7. Frissell: Jour. Am. Med. Assn., 1911, lvi, 661.

spinal fluid by simple chemical and microscopic methods must necessarily be merely relative. While in cerebrospinal meningitis, in tuberculous meningitis, in pneumococcus, influenzal and similar types of meningitis one obtains specific information and is usually enabled to make a positive diagnosis from an examination of the spinal fluid, the value of the examination in poliomyelitis is necessarily less direct. Thus in a fluid containing a high percentage of polymorphonuclear cells, the failure to find any organisms would certainly be suggestive of poliomyelitis. The difficulty of differential diagnosis is perhaps greater when one has a fluid with a lymphocytic cytology. Lucas has discussed at length the non-specificity of the cytological findings in various meningeal conditions, and finds a similar cell picture in encephalitis, poliomyelitis, meningismus, tuberculous meningitis and syphilis of the central nervous system. Because, however, the examination of the spinal fluid in poliomyelitis lacks specific value, it would be very wrong to conclude that it was not an important aid in diagnosis, or that lumbar puncture should not be performed in suspicious cases. Routine blood examinations and the usual urinary analyses have comparatively rarely any specific diagnostic value. Their greatest usefulness consists, perhaps, in helping to rule out certain possibilities, and in focusing one's attention on a narrower group of processes. Within such a group the blood or urine examination, partly of itself, and partly when brought into line with clinical experience, may, by bringing either negative or positive evidence, be the determining factor which inclines one toward the correct diagnosis. The value of positive findings in spinal fluid examinations is undoubted. The value of negative results in examination approaches it in importance. Thus in the case of poliomyelitis, the failure to demonstrate specific changes in the spinal fluid should rule out several serious meningeal conditions, the differential diagnosis of which from poliomyelitis in the preparalytic stage may be most confusing. On the other hand, by the demonstration of non-specific changes (for we have seen that by far the greater number of fluids in poliomyelitis are in one way or another abnormal), a host of other conditions which may simulate incipient poliomyelitis are removed from further consideration. Among the small group of remaining possibilities, certain characteristic types of fluid, as for instance one with a high cell count and a normal globulin reaction, may be of some value in deciding the diagnosis. More careful studies of the fluids in conditions other than poliomyelitis may throw further light on the differential value of non-specific fluids. At any rate, when even the non-specific fluid of poliomyelitis is considered in association with the clinical features of the case in hand, the diagnosis can probably be made in the great majority of cases even in the preparalytic stage. The ease and safety with which lumbar puncture can be performed and

the simplicity of the examination of spinal fluids should make the method one of much wider use than it is at present. The value of any future method of treatment of poliomyelitis must depend eventually on the possibility of early diagnosis, for where nerve cells have been destroyed, the results from any therapeutic measures will be comparatively small.

CONCLUSIONS

The spinal fluid from cases of acute poliomyelitis during the first few weeks after the onset of symptoms shows, in the great majority of instances, deviations from the normal.

Fluids taken during the early days of the disease, and especially before the onset of paralysis, tend to show an increased cell count with a low or normal globulin content.

At this early stage the polymorphonuclear cells may amount to 90 per cent. of the total. Most fluids, however, show lymphocytes and large mononuclear cells almost exclusively.

After the first two weeks the cell count usually drops to normal, or nearly to normal, and there is frequently an increase in the globulin content. A slight increase in globulin may persist for seven weeks or more.

Analogous changes may be found in the spinal fluid of abortive cases.

All fluids examined reduced Fehling's solution.

The examination of the cerebrospinal fluid in acute poliomyelitis, while giving, as far as is yet known, no specific diagnostic criteria, is of the utmost value as an aid to diagnosis both in preparalytic and in abortive cases.

THE BLOOD

The complete symptomatology of diseases caused by filterable virus in man is not yet established. Certain clinical features of the two most studied examples, hydrophobia and poliomyelitis, have been well described, but the blood picture which has been so thoroughly determined in most infectious diseases and is of such value in differential diagnosis, is little discussed. Owing to the resemblance of numerous features of rabies and poliomyelitis, it seemed that a comparative study of the blood changes in the two diseases might throw some further light on the nature of the human body's reaction to invasion by infecting agents of this type. The large systems of medicine make no mention of blood counts in either malady. In one exhaustive report of thirty cases of rabies by Brain and Maloney the blood of one patient is reported to have had a "slight polymorphonuclear leukocytosis."

Through the courtesy of the attending physicians at the Presbyterian Hospital, in New York, the following blood counts from cases of hydrophobia are available:

Patient 1.—Male, aged 35; temperature, 99.3 F.; W. B. C., 9,400; polymorphonuclears, 76.0 per cent.; lymphocytes, 10.5 per cent.; large mononuclears, 9.8 per cent.; transitionals, 3.8 per cent.; basophils, 0.0 per cent.; eosinophils, 0.0 per cent.

Patient 2.—Male, aged 27; temperature, 100 to 103 F.; W. B. C., 13,000: Polymorphonuclears, 76.5 per cent.; lymphocytes, 11.0 per cent.; large mononuclears, 10.0 per cent.; transitionals, 2.5 per cent.; basophils, 0.0 per cent.; eosinophils, 0.0 per cent.

Patient 3.—Male, aged 27; temperature, 103 F. (Nov. 26). W. B. C., 34,450: Polymorphonuclears, 85.0 per cent.; lymphocytes, 12.0; large mononuclears, 0.0 per cent.; transitionals, 0.3 per cent.; basophils, 0.0 per cent.; eosinophils, 0.0 per cent.

Patient 3.—Temperature 103 F. (Nov. 27). W. B. C., 30,200: Polymorphonuclears, 87.0 per cent.; lymphocytes, 10.5 per cent.; large mononuclears, 0.5 per cent.; transitionals, 2.0 per cent.; basophils, 0.0 per cent.; eosinophils, 0.0 per cent.

Obviously, except in the first case, in which the total count was normal, there was a leukocytosis. In all the cases a definite polynucleosis existed with low lymphocyte percentage.

In the literature of poliomyelitis there are conflicting statements about the leukocyte count. LaFetra[8] in New York and Müller[9] in Germany have made the most extensive observations on the blood. The former reported in six cases a leukocytosis, running from 13,400 to 20,600. He says nothing about the differential counts. Müller, on the other hand, reporting fifteen cases, says that a leukopenia of from 3,000 to 5,000 was always present in the acute stage. He gives no figures but states that there was a slight increase of the lymphocytes. Gay and Lucas[10] made a comparative study of the blood in monkeys suffering from the experimentally produced disease and in children with poliomyelitis. In monkeys they report a leukopenia during the acute stage with a lymphocytosis ranging from 10 to 20 per cent. above normal. The protocols of the human cases, however, do not offer very convincing support to their statements that a similar blood picture occurs in the human cases. The lowest count reported from a 9 months' baby (Case 2), is 7,800 to 12,200. The other three cases showed the following leukocyte counts:

Case 1, 20 months, 11,600 to 17,400.
Case 3, 2 years, 12,000.
Case 4, 5 years, 13,400.

These blood counts can hardly be considered below normal. In two of their four cases a lymphocytosis, "slight" for one and "62 per cent." for the other, is given. Of the other two cases, one, a child of 5 years, had a normal differential count and the other a polynucleosis of 75.5 per cent.

The accompanying tables and ensuing statements are based on the study of the blood of our seventy-one hospital patients. The actual figures given represent the counts from only fifty-nine cases. The records

8. La Fetra: Arch. Pediat., 1909, xxvi, 328.
9. Müller: Die Spinale Kinderlähmung, Berlin, 1910.
10. Gay and Lucas: Arch. Int. Med., 1910, vi, 330.

of the other twelve cases were omitted because those patients suffered from other conditions which might have affected the blood count. Care was taken to avoid collecting the blood at times when a digestive leukocytosis might have been present. The total white count was made in the usual way, the blood and 0.5 acetic acid being mixed in a 1-10 pipette and the drop counted on a 0.1 mm. cell. For differential counts Wright's modification of the Romanowski stain was used.

TABLE 1.—DIFFERENTIAL BLOOD-COUNT IN CASES OF ACUTE POLIOMYELITIS

Age, Years	Number of Cases			Poly.	Lymph.	L.Mono.	Trans.	Baso.	Eosin.	Stim.*
			First Week							
½-1	4	23,900-35,600	Max.	59.0	57.0	18.0	15.0	0.5	1.0	1.5
			Min.	25.0	16.5	1.0	0.5	0.0	0.0	0.0
			Aver.	42.0	36.7	9.5	7.7	....	....	....
1-2	12	12,200-26,000	Max.	70.0	50.0	13.5	13.5	1.0	15.5	1.0
			Min.	26.5	16.5	1.5	2.5	0.0	0.0	0.0
			Aver.	48.2	33.2	7.5	8.0	....	....	....
2-3	5	9,000-23,000	Max.	64.5	43.0	14.0	18.0	1.5	7.0	1.5
			Min.	32.5	20.0	1.5	3.0	0.0	0.0	0.0
			Aver.	48.5	31.5	7.7	10.5	....	....	....
3-4	4	6,900-15,000	Max.	72.0	29.0	8.5	13.0	0.5	9.0	1.0
			Min.	55.0	17.0	1.0	1.5	0.0	0.0	0.0
			Aver.	68.5	23.0	4.7	7.2	....	....	....
4-5	6	12,400-33,000	Max.	81.0	37.5	26.5	14.5	1.5	0.0	0.5
			Min.	44.5	18.6	2.6	4.5	0.0	0.0	0.0
			Aver.	62.7	28.0	14.5	9.5	....	....	....
5-7	4	8,800-23,300	Max.	77.0	24.0	11.7	5.5	1.0	8.0	1.0
			Min.	62.2	16.7	0.5	1.0	0.0	0.0	0.0
			Aver.	69.6	20.3	6.5	3.2	....	....	....
8-21	3	12,000-16,700	Max.	75.0	17.5	17.0	9.5	0.05	1.5	0.0
	—		Min.	59.0	13.0	0.5	4.5	0.0	0.0	0.0
	38		Aver.	67.0	15.2	8.7	7.0	....	....	....
			Second Week.							
1-2	11	12,300-30,400	Max.	73.0	56.0	23.0	11.0	6.0	5.5	0.05
			Min.	37.0	9.0	0.5	4.0	0.0	0.0	0.0
			Aver.	55.0	32.5	11.7	7.7	....	....	....
2-3	8	9,800-20,000	Max.	61.5	41.0	23.5	10.5	5.0	7.5	0.5
			Min.	38.0	10.0	2.0	2.0	0.0	0.0	0.0
			Aver.	49.7	25.5	12.7	6.2	....	....	....
3-4	2	14,600-19,700	Max.	84.0	21.5	13.5	6.5	0.0	0.5	0.5
			Min.	57.5	6.5	6.5	3.0	0.0	0.0	0.0
			Aver.	70.7	14.0	10.0	4.7	....	....	....
4-5	1	28,000								
5-7	1	11,800		76.5	14.5	4.5	3.5	0.0	0.5	0.5
8-12	1	12,000		62.0	17.5	16.0	3.5	0.0	1.0	0.0
	—									
	24									

*Stim="Stimulation Forms" of Türck.

Age, Years	Number of Cases			Poly.	Lymph.	L.Mono.	Trans.	Baso.	Eosin.	Stim.
				Third Week						
			Max.	67.0	58.5	7.5	13.5	1.5	10.5	1.0
1-2	13	8,300-26,100	Min.	30.5	22.0	0.0	0.5	0.0	0.0	0.0
			Aver.	48.7	40.2	3.7	7.0	....	....	....
			Max.	63.5	42.5	24.5	9.5	0.5	3.0	0.5
2-3	5	12,800-23,700	Min.	45.0	16.0	0.0	2.5	0.0	1.0	0.0
			Aver.	54.7	29.2	12.2	6.0	....	....	....
			Max.	59.5	37.5	6.5	14.0	1.0	6.5	0.0
3-4	2	10,300-15,700	Min.	45.0	20.0	2.0	5.5	0.0	0.5	0.0
			Aver.	52.2	28.7	4.2	9.7	....	....	....
4-5	1	28,000								
5-7	1	28,000		72.0	9.0	2.0	10.0	0.0	2.0	0.0
			Max.	67.5	28.0	4.5	9.0	0.0	2.5	0.0
8-12	2	7,800-18,000	Min.	63.0	19.5	3.5	2.0	0.0	1.0	0.0
	24		Aver.	65.2	23.7	4.0	....	....	....	....
				Fourth Week						
			Max.	60.0	63.5	41.0	17.0	1.0	4.0	2.5
1-2	11	10,100-24,700	Min.	26.5	21.5	35.0	1.5	0.0	0.0	0.0
			Aver.	43.2	42.5	23.7	9.5	....	....	....
			Max.	65.0	46.0	10.5	10.0	0.5	6.0	0.0
2-3	5	14,600-23,600	Min.	26.5	23.5	0.5	4.5	0.0	0.0	0.0
			Aver.	45.7	34.7	5.5	7.2	....	....	....
			Max.	51.0	46.5	1.5	8.5	0.5	8.0	0.5
3-4	2	11,000-19,800	Min.	40.5	37.5	0.0	3.5	0.0	2.0	0.0
			Aver.	45.7	42.0	0.7	6.0	....	....	....
			Max.	58.5	33.8	10.5	4.0	0.6	2.7	0.3
4-5	3	10,500-22,300	Min.	42.0	27.9	6.4	2.0	0.0	0.5	0.0
			Aver.	50.2	30.8	8.4	3.0	....	....	....
			Max.	64.5	36.0	12.5	2.5	0.5	0.0	0.0
5-7	2	14,200-21,000	Min.	49.5	25.0	7.5	1.5	0.0	0.0	0.0
			Aver.	57.0	30.0	10.0	2.0	....	....	....
			Max.	64.5	26.0	2.5	8.0	0.5	4.5	0.5
8-12	2	9,400-10,700	Min.	62.5	....	0.0	4.5	0.0	0.5	0.0
	25		Aver.	63.5	....	1.7	6.2	....	....	....
				Fifth Week						
			Max.	60.5	56.0	13.5	10.0	0.5	6.5	1.0
1-2	6	9,100-23,600	Minn.	38.0	20.5	1.0	2.0	0.0	1.5	0.0
			Aver.	49.2	38.2	7.2	6.0	....	....	....
			Max.	58.0	44.5	9.5	14.0	0.0	9.5	0.5
2-3	6	15,000-27,000	Min.	40.0	21.5	0.0	5.0	0.0	3.5	0.0
			Aver.	49.0	33.0	4.2	9.5	....	....	....
			Max.	60.5	30.0	10.5	6.5	1.0	4.0	0.5
3-4	3	12,800-16,700	Min.	54.5	25.0	6.0	2.5	0.0	2.0	0.0
			Aver.	57.5	27.5	8.2	4.5	....	....	....
			Max.	85.0	24.0	12.0	7.0	1.0	2.0	1.0
4-5	2	6,300-20,000	Min.	62.5	19.0	0.5	3.5	0.0	....	0.0
	17		Aver.	73.7	21.5	6.2	5.2	....	....	....

Sixth Week

Age, Years	Number of Cases			Poly.	Lymph.	L.Mono.	Trans.	Baso.	Eosin.	Stim.
1-2	3	9,600-26,700	Max.	40.0	69.0	10.0	15.0	0.5	2.5	2.0
			Min.	23.0	32.0	0.0	0.5	0.0	0.5	0.0
			Aver.	31.5	50.5	5.0	7.7	....	....	....
2-3	2	10,200-22,800	Max.	57.0	44.0	4.5	9.0	0.5	3.5	2.0
			Min.	45.0	29.0	2.0	0.5	0.0	0.5	0.5
			Aver.	51.0	36.5	3.2	4.7	....	....	....
4-5	2	13,100-28,600	Max.	64.0	34.0	8.5	3.0	0.0	1.0	0.0
			Min.	57.5	23.5	7.0	1.5	0.0	0.0	0.0
			Aver.	60.7	28.7	7.7	2.2	....	....	....
5-7	1	15,000		50.0	32.5	4.0	11.0	0.0	1.5	1.0
8-12	1	9,400		47.0	37.5	5.5	4.0	0.0	3.5	2.5
	9									

Seventh Week

1-2	2	23,700-26,700	Max.	51.0	45.0	10.0	5.0	2.0	6.5	0.5
			Min.	31.5	34.5	9.5	4.0	0.0	0.5	0.0
			Aver.	41.2	39.7	9.7	....	....	....	....
2-3	3	15,800-22,300	Max.	70.0	43.6	11.0	7.5	0.5	5.0	0.0
			Min.	42.5	16.0	0.5	3.5	0.0	1.0	0.0
			Aver.	56.2	29.8	5.7	5.5	....	....	....
3-4	1	9,800		49.0	30.5	4.0	11.5	0.5	4.0	0.0
4-5	2	11,000-12,400	Max.	62.0	54.5	10.5	4.0	9.5	1.0	0.5
			Min.	39.5	16.0	0.5	0.5	0.5	0.5	....
			Aver.	50.7	35.2	5.5	2.2	....	....	....
5-7	1	16,000		59.5	22.0	7.5	8.5	0.5	2.0	0.0
	9									

Eighth Week

4-5	2	10,700-12,000	Max.	52.5	37.5	9.0	5.5	0.0	1.5	0.0
			Min.	47.0	35.5	8.5	2.5	0.0	0.5	....
			Aver.	49.7	36.5	8.7	4.0	....	....	....
8-12	1	16,000		58.0	32.5	2.0	6.0	0.0	2.0	0.5
	3									

Tenth Week

4-5	1	10,800		61.5	23.5	4.5	9.0	0.0	0.0	0.5

Twelfth Week

4-5	1	16,000		54.0	38.0	5.5	2.0	0.0	1.5	0.0

The first set of tables is designed to show minimal, maximal and average counts for groups of cases. A second, composite table shows minimal, maximal and average total leukocyte counts of all ages together by weeks. Such a compilation seems justifiable because the age variation is much less in respect to the total than the differential leukocyte count.

TABLE 2.—COMPOSITE TABLE SHOWING MINIMAL, MAXIMAL AND AVERAGE TOTAL LEUKOCYTE COUNTS OF ALL AGES BY WEEKS

Week	Number of Cases	Maximal	Minimal	Average
First	38	24,600	12,200	18,400
Second	24	24,500	12,000	18,250
Third	24	23,300	10,400	16,800
Fourth	25	20,300	11,600	15,900
Fifth	17	21,800	10,800	16,300
Sixth	9	23,200	10,500	16,800
Seventh	9	19,500	15,000	17,250
Eighth	3	14,000	10,700	12,400
Tenth	1	.....	.....	10,800
Twelfth	1	.....	.....	16,000
	151			

TABLE 3.—THIS TABLE COMPRISES SEPARATELY THE COUNTS FROM PREPARALYTIC AND ABORTIVE CASES

Case	Age, Yrs.		W. B. C.	Poly.	Lymph.	L.Mono.	Trans.	Baso.	Eosin.	Stim.
B. X.	5		15,000	....	....	....	....	....	....	....
		3/0*	7,000	81.0	....	....	....	....	....	....
J. S.	2½	2/0	14,200	58.5	31.5	7.0	3.0	0.0	0.0	0.0
C. T.	⅔	2/0	35,600	32.0	55.0	1.0	9.0	0.0	1.0	1.5
E. N.	6	6/0	23,200	76.0	17.0	3.0	2.5	0.0	1.0	0.5
H. B.	6	2/0	8,800	77.0	17.5	0.5	4.0	0.0	0.0	1.0
		3/0	11,200	79.5	14.5	0.5	4.5	0.0	0.5	0.5
		6/0	5,600	68.5	22.0	1.5	6.0	0.0	1.0	1.0
		13/0	11,800	76.5	14.5	4.5	3.5	0.0	0.5	0.5
P. T.	2	1/0	15,000	52.5	31.5	13.5	1.5	0.5	0.5	0.0

*The numerator of the fraction signifies the day of the disease, the denominator the day of the paralysis.

It is obvious from these figures that while in most cases in the preparalytic stage the total leukocyte count varies within the normal, there is a tendency toward the upper limits. More cases showed counts well above than below the usual extremes. In the differential counts there is a definite polynucleosis with the one exception of an 8 months' infant in whom the relation of polymorphonuclears and lymphocytes is normal. Furthermore, the lymphocyte percentages in all the other cases are distinctly below the usual figure.

TABLE 4.—DIFFERENTIAL COUNTS IN NORMAL INDIVIDUALS

Age, Years		Poly.	Lymph.	L.Mono.	Eosin.	Baso.
	Max.	33.9	58.5	12.2	4.5	0.8
½-1	Min.	24.6	50.5	7.3	0.0	0.1
	Aver.	30.4	55.9	9.6	2.6	0.4
	Max.	39.7	58.8	11.7	6.0	0.5
1-2	Min.	27.5	45.3	6.7	1.6	0.0
	Aver.	36.3	51.2	8.5	3.2	0.2
	Max.	44.3	55.0	11.3	6.0	1.2
2-3	Minn.	33.2	43.5	5.0	0.5	0.0
	Aver.	38.7	49.0	8.2	3.1	0.4
	Max.	54.1	47.6	16.2	4.2	0.9
3-4	Min.	36.2	32.2	5.0	1.5	0.0
	Aver.	44.7	39.1	11.2	2.8	0.5
	Max.	51.7	49.5	6.7	4.0	0.6
4-5	Min.	42.2	38.4	3.4	1.6	0.3
	Aver.	48.5	42.1	6.0	2.6	0.3
	Max.	61.8	36.7	16.0	4.7	1.0
5-6	Min.	52.6	21.2	6.5	0.7	0.3
	Aver.	56.5	29.9	10.0	2.5	0.6
	Max.	61.3	34.1	15.7	4.7	0.6
6-7	Min.	52.3	24.5	8.1	0.1	0.0
	Aver.	56.0	30.4	10.8	2.2	0.2
	Max.	72.0	39.1	15.2	3.5	0.2
7-8	Min.	45.2	21.1	6.7	0.0	0.0
	Aver.	54.4	32.5	11.6	0.1	0.06

In one instance in which blood counts were made by the physician before the patient came to the hospital, a low total count appeared, 7,000 two days before the paralysis and 3,000 on the day after paralysis. The differential count, however, showed a polymorphonuclear increase each time (81 per cent. and 58 per cent). Except for this one case which subsequently became one of our seventy-one, we have not seen a true leukopenia. One abortive case in a little girl 6 years of age showed counts of 5,600 to 11,800. In the youngest children the leukocyte counts have been highest, and the polymorphonuclear increase has been both relatively and actually more marked than in the older patients. A glance at the tables, however, will show that all ages have a high white cell count. Furthermore, the leukocytosis persists for a surprising length of time. In nine cases, for example, observed during the seventh week, the leukocytes numbered 15,000 to 19,500 with an average of 17,250.

The differential count presents a fairly constant picture. For comparison, a table of differential counts from normal individuals prepared

by Schloss[11] is printed. There is obviously in poliomyelitis an increase in polymorphonuclear cells of 10 to 15 per cent. above the normal. This increase appears irrespective of the patient's age, though the youngest children have shown perhaps the highest polynuclear counts. Contrary to the observations of Müller and Gay and Lucas, we have found a diminution of lymphocytes. In general, they have been from 15 to 20 per cent. below the normal percentages. Notwithstanding, however, the great variation which these cells present in relation to age, the finding of low lymphocyte percentages has been constant in all our cases of poliomyelitis.

No abnormality was noted in the number of transitional and large mononuclear cells. In a good many cases there has been an increase in eosinophils. This finding, however, has not been sufficiently constant to justify further comment. No counts were made of the red cells, nor were any hemoglobin estimations recorded. There has been nothing, however, in the appearance of the patients to suggest an anemia resulting from the disease; nor did the stained erythrocytes look abnormal. No actual counts were made of blood platelets, but the impression was gained in the course of many differential counts that these elements were somewhat increased.

To sum up, then, we have found in the blood of patients with poliomyelitis a constant and marked leukocytosis. In several instances the count has been as high as 30,000. In only one case has there been a definite leukopenia. Besides the increase in total cells, there has been an equally constant increase of polymorphonuclears of 10 to 15 per cent. above the normal, and a diminution of lymphocytes of 15 to 20 per cent. The other forms of leukocytes have shown no abnormalities.

What the significance of this leukocytosis, and especially the polynucleosis, may be, is at present not clear. In view of the fact that the brunt of the body's attempt at defense falls on the lymphatic system, and that the cells which are marshalled at the point of attack seem to be lymphocytes, one might be led at first thought to expect a lymphocytosis in the blood. On the other hand, it is conceivable that the lymphocytes are relatively few in the circulation because they are constantly being withdrawn to meet the invasion of the virus in the spinal cord. It will require, however, a more fundamental knowledge of the kind of reaction the body develops to this type of infecting agent before a correct explanation of the blood picture can be made. At all events, while the blood picture in poliomyelitis is perhaps not any more specific than are the changes in the spinal fluid, it is at least helpful. If taken in connection with other available evidence, a leukocytosis of 15,000 to 30,000 is

11. Schloss: "The Normal Percentages of the Different Varieties of Leukocytes in Infants and Children," Arch. Int. Med., 1910, vi, 638.

distinctly suggestive of the disease in question, especially if the polymorphonuclear cells are increased at the expense of the lymphocytes.

NOTE.—Since this paper has reached the proof, certain additions to our knowledge of the pathology of poliomyelitis have appeared which indicate definitely the general systemic nature of the infection. In view of the finding in many viscera of areas of focal necrosis with many polynuclear phagocytes, an explanation for the polymorphonuclear cytosis in the blood is at once suggested. Furthermore, the appearance of many disintegrating lymphocytes lends support to the idea that rapid destruction of lymphocytes underlies the lymphopenia. Both these explanations for the blood picture are offered purely as hypotheses.

Sixty-Sixth Street and Avenue A.

ATROPHY OF INFANTS*

PROFESSOR AD. CZERNY
STRASSBURG

The term "atrophy of infants" was formerly used to designate a disease *sui generis*. It was characterized clinically by the fact that children, who had developed well up to a certain time, gradually began to fail and eventually died, presenting the picture of a progressive and often extreme grade of emaciation. This peculiar condition, which was seen only in infants, came to be known under different names, such as tabes mesenterica, athrepsia, pedatrophy and "decomposition." But the change in name brought with it no scientific or practical advance. The nutritional disturbances stand out so prominently in the clinical picture of the disease that they generally came to be recognized as the most essential feature. When the children presented a greater or less degree of atrophy, infectious processes were likely to make their appearance in various parts of the body and these hastened the fatal termination of the atrophy.

The pathologic-anatomic findings appeared to justify the assumption that the atrophy was a disease entity. The autopsies showed not only a general emaciation, but it was believed, in addition, that there was an actual atrophy of the intestinal tract. The intestinal musculature appeared to be thinned out to a marked degree and the intestinal glands to be hypotrophic. Therefore it appeared reasonable to believe that as a result of this, the nutrition of the child must have suffered and eventually could not be maintained at a point at which life was possible. More recent investigations have shown, however, that the views that have been held in regard to the atrophy of the intestinal tract are no longer tenable; that the autopsy findings have been misinterpreted, in that what appeared to have been a thinning of the intestinal wall due to atrophy, is in reality a thinning out of the muscular layers due to distention, and the histologic findings in the intestinal mucosa are directly referable to post-mortem changes. An examination of the intestinal mucosa of a child that has died as a result of atrophy shows that it is absolutely normal, provided the examination is made soon after death and with all the necessary precautions. At the present time, therefore, the pathologist can regard this atrophy only as the result of a development, far behind what it should be, and of an extreme emaciation.

* Translation by F. C. Zapffe, M.D., Chicago, of a paper read by the author at Strassburg, Nov. 7, 1911.

This unsatisfactory state of our knowledge has led to the laying of altogether too much importance on the secondary infections of atrophic children. This is especially true of otitis media. As a matter of fact, pus is often found in the middle ear of children who have died in a condition of atrophy. A natural result of this finding was the belief that bacterial products of a poisonous kind could be absorbed from the purulent areas produced by these bacteria, and that in consequence the atrophy is nothing more than the result of a septic intoxication. The question was never asked of the clinician whether the otitis media first appeared when the child was already atrophic, or whether it ushered in the entire process. The finding of a purulent otitis media was regarded as sufficient ground for teaching physicians that atrophy is the result of otitis media.

One thing at least can be said for pathologists: they were at all times of one mind. They were at first all fully convinced that there was an intestinal atrophy and they were later convinced that otitis media played a most alarming part in the high infant mortality. This unanimity of opinion was not to be found among the clinicians at any time. There are so many views held as to the nature of atrophy that I cannot, at this time, refer to them all. I will content myself with mentioning those definite facts to which we can hold fast and what outlook these allow for medical activity.

Atrophy is not a disease *per se,* but a disturbance in growth and in general nutrition, which is the result of nutritional disturbances, or of infectious processes, or of both. The clinical picture may be the same in all these instances, but a differentiation based on etiology is of great importance to us on account of prophylaxis and therapy. If the disease is a nutritional disturbance, splendid results can be obtained by dietetic management. If, however, an infection, which we cannot therapeutically influence, is the exciting factor, we are often placed in a position where we can neither check nor cure the atrophy.

Before going deeper into the subject, I should like to put the question before you, whether under like conditions every nursing infant becomes atrophic or whether to do so is the tendency of only certain ones. And I should like to answer this question by saying that the occurrence of atrophy, irrespective of the causes producing it, is distinctly the result of a constitutional anomaly. We are all aware that the same errors are made in feeding many children, and yet only a proportion of these become atrophic. The same difference obtains with children suffering from infectious processes. For instance, a child may pass safely through an attack of whooping-cough, bronchopneumonia or otitis media without any severe influence on his general condition or his growth being apparent, whereas the general condition of other children may be affected

in the severest way by these same diseases. Naturally, in the case of infections one thinks of a different degree of virulence of the organisms. But even when there may be actually a variation in virulence this does not by any manner of means explain the great difference in the effect on the general condition and the growth of the child.

Every clinician, and still better, every family physician, knows that there are many families in which an infant, as the result of a nutritional disturbance or of an infection, will temporarily lose weight, but in which a case of atrophy never develops. While on the other hand, there are families in which even under good conditions, one or more infants pass through some stage of atrophy. These facts are of the very greatest importance, because the majority of physicians have been rather inclined to accept the statement that atrophy is always the result of poor nutrition, deficient care and poor hygiene. Of course, it cannot be denied that all these factors may greatly influence the course of the atrophy; yet everything is not to be explained as a result of them, because we continually see children who do not become atrophic in spite of every improper and dangerous kind of food. If we restrict our studies entirely to the children of the working class, we hardly obtain information as to the fundamental character of atrophy, for in this class it is seldom possible to obtain as exact a history as is necessary to clear up the question or to exclude the non-essentials. However, as I have previously stated, this condition of atrophy in infants is seen not only in children of the lower classes, but also in those better situated, and it is in these that one is able to obtain a more exact idea of the essential process of the disease.

We may differentiate three classes of children that may suffer from this condition more or less during infancy. The first group comprises children that have inherited the exudative diathesis (Exudative Diatheses, Czerny). The milder forms are recognized only by the difficulties in the way of proper nutrition of the child. Children with a more severe form of exudative diathesis readily become atrophic if the diet is not very carefully regulated. It is especially instructive that this condition also occurs in breast-fed infants. The majority of these children may be recognized from the very beginning by the fact that, irrespective of whether they are nursed by their mothers or by wet-nurses, they regain their birth weights late and also that they do not show the development and increase in weight which is characteristic of normal children under similar conditions. If, in severe cases, breast milk is the only food given, on the basis that it is the best food for all children, the complete clinical picture of atrophy may develop. This takes place the more readily because children with the exudative diathesis are susceptible to infections and are hindered in their development by such to a most marked degree. Continued study of this atrophy as a result of an exudative diathesis has convinced me

that its development in the beginning is favored by the injurious effect of improper food. A change from a milk rich in fat to an artificial food containing much carbohydrate and little fat, brings about a cure of the atrophy. If the diet is regulated so that these children do not become too fat or retain too much water, the disposition to infection, which is characteristic of the exudative diathesis, becomes less.

Among the better classes the children receive attention more promptly and medical advice is sought early, so that it is relatively seldom that severe grades of atrophy occur. Among the lower classes the mother usually experiments with the diet for a while herself, or she seeks the advice of other women, especially midwives, whose knowledge for such cases is insufficient, and it is on this account that one can see in children's hospitals and infants' homes a large number of children with exudative diathesis in the atrophic stage. The cases are not thankless for therapeutics; and examples of cure furnish the chief objects of advertisement for children's departments.

The second class or group of children who readily fall victims to atrophy, are those of neuropathic and psychopathic parents. They offer much greater difficulty in the way of treatment and the results under even the most favorable circumstances are often very unsatisfactory. The extreme irritability of the nervous system of the intestinal tract and certain psychic peculiarities furnish the foundation for the atrophy. I should not wish, however, to be misunderstood as maintaining that every child of psychoneuropathic parents must present the same pathologic qualities. Such children inherit sometimes an irritability of one part of the nervous system and sometimes of another, and only one group of children in this class is in danger of becoming atrophic during infancy. The excessive irritability of the intestinal nervous mechanism is manifested in the main by the fact that these children vomit on slight provocation. The vomiting is sometimes so severe that it is almost impossible to administer sufficient food, so long as a fluid diet is necessary. When a soft diet can be given in place of the fluid diet, the vomiting ceases. Sufficient time may have elapsed, however, to have produced an atrophy, the result of insufficient nourishment.

So far as the intestines are concerned, this state of the nervous system shows itself by the fact that such children react to irritation in a way that normal children would not, and have numerous stools, and because numerous, of an abnormal character. As a result of this, we see that even the unavoidable variations in the composition of the mother's milk may produce diarrhea. The result is the same with artificial feeding. These children react with diarrhea to a relatively small amount of carbohydrate in the diet. As a certain amount of carbohydrate in the food is an absolute necessity for the welfare of a child, and especially for a

gain in weight, those who receive the carefully regulated quantity of carbohydrate necessary remain backward in weight and become thereby more or less atrophic. When certain psychic anomalies are present the difficulties of the situation are manifestly increased. The children of neuropathic parents often have the trait of persistently refusing articles with a taste to which normal children readily become accustomed. The difficulty is then that the child so positively refuses the food that we have ordered that he can hardly be made to take it. Unfortunately, instinct does not induce these children to take only such food as is beneficial to them and to refuse that which could be injurious. They often drink greedily a food as a result of which they have plainly become atrophic, and energetically refuse for days the taking of a food by which they eventually will be cured. The treatment of such children always requires a wide experience, and the exercise of great patience and energy. These factors are not always obtainable and therefore it is not astonishing that such children often present severe grades of atrophy even under favorable circumstances.

It should also be mentioned that these psychopathic children are almost always to be recognized by their bad sleeping and great restlessness. They respond to stimuli which the normal child would not notice and often with evidences of dislike. This circumstance increases the danger of atrophy as a result of alimentary causes, because those responsible for children may feel called on, on account of the restlessness, to experiment with the food and do not always do this intelligently and successfully.

From what has been said it is sufficiently apparent that in the case of many children of psychoneuropathic parents, there are numerous factors that may make proper and sufficient feeding difficult or almost impossible. Most of the children seen in clinics and in institutions with whom unsatisfactory results are obtained, belong to this class. And still it is not infrequently possible with proper care to keep them alive. A study of the death-rate of infants shows that this type contributes largely to the mortality. This is not solely the result of the difficulties mentioned but because in such children there are still other anomalies of innervation and these have very bad results especially if they affect the circulation.

In the third class or group are the children with the "hydropic" constitution who in consequence of disturbances of nutrition readily develop atrophy. Children with such a tendency show differences in salt and water retention from normal children. These children retain readily large quantities of water in their tissues. However, the bond between the water and the salts, the carbohydrates and the colloidal substances in the tissues must be much weaker than under normal conditions because

the slightest nutritional disturbance causes wide variations in the water content of the body. This is shown, even when no disturbances are present, by the great variations in the daily body weight. Children of this group will readily gain a great deal of weight when a diet rich in sugars and salt is given. Acute nutritional disturbances and infectious diseases cause such an extreme loss of fluid as to lead to an atrophy which is recovered from with difficulty.

It is quite evident from what has been said, that atrophy may result from many different causes. In the one instance, a child with exudative diathesis may become atrophic as the result of a food rich in fat; in another instance, a neuropathic child may reach apparently the same condition in consequence of an insufficient quantity of food, and in the third case, the lability of the water content of the organism is responsible for similar results.

A consideration of the constitution of children makes it clear to us that there are certain prerequisite conditions that must be present in the body in order that atrophy may develop, but this gives us no clear idea as to the pathogenesis of the condition. Atrophy is characterized by a cessation of normal growth in the entire organism. This disturbance in growth manifests itself in one direction by the absence of increase in body weight or even by a loss. This is easily understood because in such cases the development of fat ceases. Emaciation of varying degree is the external evidence of this. The atrophic child does not grow nor does he accumulate fat even when given a food which under normal conditions would lead to an increase in body weight and increase in fat. If one is inclined to the belief that the absence of fat is caused by a lack of development of adipose tissue, one may say that atrophy is a disturbance of growth which is most manifest in the lack of development of adipose tissue.

Both the failure of general growth and of fat formation occasioned by atrophy are, however, fully reparable. As soon as the nutritional disturbance is overcome or the infection ceases, growth proceeds in the normal manner and the delay is compensated for. We must conclude, therefore, that the disturbance is not a deep-seated one in the cell-complex of the infant's organism. The nature of the injury has not been positively ascertained. From comparison with the etiology of disturbances of plant physiology we may assume that inorganic substances play a considerable part in the production of atrophy. I cite, for instance, the striking influence that potassium exerts on the growth of plants. In the acute nutritional disturbances of infants the organism loses, according to published observations, a particularly large amount of potassium. If potassium exerts an effect on animal cells similar to that on plant cells, then the failure of growth in atrophy is understandable. But the condi-

tions are apparently not as simple in the human organism. It would be, at the present time, very presumptuous to ascribe the disturbances of growth in atrophy to the loss of a single mineral substance. But it is in this way possible for us to conceive how apparently dissimilar causes may give a similar clinical picture of atrophy. They all lead to the loss of mineral substances and apparently chiefly of those which are of determining influence for the growth of the cells.

Similar disturbances are also seen in the case of older children and yet in these, the clinical picture of atrophy is not easily produced. As I have mentioned before, infants living under like conditions do not all become atrophic. To explain this difference we must consider the facts which at present are known to govern the growth of the infant and those which govern the growth of the older child. We know that in the case of the former, it is not exclusively the material furnished in the food which is responsible for growth but that certain deposits in the body are drawn on and that these are present at the time of birth and are gradually used up during the lactation period. Under normal conditions these deposits suffice to compensate for a greater or less loss, the result of pathologic processes. In the case of the child who is hereditarily affected so that one or the other of these depots is deficient, even at birth, consequences of many kinds follow any disturbance that may arise, and one of these consequences is atrophy. That this is not a consideration of mere theoretical value is seen from the fact that what theoretically should cure the atrophy, actually does so. It is only with difficulty and in the less severe grades of atrophy that an exclusively milk diet, even of mother's milk, can produce a cure. In the severe cases this is absolutely impossible. Atrophy requires a food in which, in addition to the milk, certain components should be present in order to compensate for the loss that has taken place as a result of disease. The choice of the substances to be added must be made not alone with the idea of compensation, but it must also, in those cases where disturbances of nutrition have been the determining factor, be one which prevents the return of these.

With this idea in mind the numerous therapeutic measures that are employed have come into use, but at the present time they have more empirical than scientifically proved data to recommend them. At the present time only so much can be said in regard to the food components, that it is the fat that must be much restricted in the treatment of atrophy and that the place of this must be taken by carbohydrates.

PROGRESS IN PEDIATRICS

RÉSUMÉ OF WORK ON TUBERCULOSIS IN CHILDREN

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MODE OF INFECTION

1. Potter, P. S.: The Portals of Entry of Tuberculosis in Children. Pediatrics, 1911, xxiii, No. 8.

2. Cobbett, L.: Portals of Entry in Tuberculosis. Brit. Jour. Child. Dis., 1911, viii, 415.

3. Whipman, T. R.: The Portals of Infection in Tuberculosis. Brit. Jour. Child. Dis., 1910, vii, 350.

4. Leuenberger, G.: Contribution to Placental and Congenital Tuberculosis. Beitr. z. Geburtsh. u. Gynäk., 1909-10, v.

5. Gergely, E.: Cases of Congenital Tuberculosis. Zentralb. f. Kinderh., 1910, 321.

6. Peisich, K.: Tuberculosis in Nurslings and Children. Wien med. Wchnschr., 1911, Nos. 4-5, 190.

7. Moeller, A.: Hygiene of the Mouth and Pulmonary Tuberculosis. München. med. Wchnschr., 1910, No. 2.

8. Ehrhardt, O.: Tuberculosis of the Mucous Membrane of the Mouth and Lower Jaw, Following Extraction of a Tooth. Zentralb. f. Kinderh., 1911, xvi, No. 5.

9. Mathews, F. S.: Relation of Tonsil to Tubercular Adenitis. Med. Rec. New York, 1910, ii, 515.

10. Simon, G.: Adenoid Growths and their Relation to Tuberculosis, Beitr. z. Klin. d. Tuberk., 1911, xix, No. 2.

11. Warden. C. C.: Relation between the Tonsils and Tubercle Bacilli, California State Jour. Med., 1911, p. 249.

P. S. Potter[1] classifies the portals of entry of the tubercle bacilli into the human organism: (1) Prenatal infection. This may occur in three ways: (*a*) germinal transmission, (*b*) placental transmission, (*c*) intrapartum transmission. The possibility of the seed carrying the disease from the father is demonstrated by Freedman, who injected into the vagina of guinea-pigs after coitus a solution containing bacilli and a week later killed the pigs. He found the bacilli lying in the embryonic layer. (2) Infection through the buccal and nasopharyngeal mucous membranes. (3) Infection through the skin. (4) Aerogenic infection. (5) Enterogenic infection.

After a careful perusal of the literature Potter concludes that any part may serve at some time as the portal of entrance of the tubercle bacillus, but in the majority of instances it is the respiratory or alimentary tract.

L. Cobbett[2] believes that infection in phthisis comes chiefly through the mucous membrane of the respiratory tract. He criticizes the theory of Calmette and the Lille school, that it results from ingestion of tubercle bacilli. This conclusion is based on the possibility of producing phthisis by feeding animals tubercle bacilli through a tube; and on experiments of Vansteenberghe and Grysez, who found anthracosis of the lungs after feeding guinea-pigs india ink and carbon. Cobbett believes the anthracosis was the natural anthracosis of the city-bred guinea-pig. He failed to find any trace of anthracosis after feeding india ink to country-bred guinea-pigs.

In feeding experiments with animals there is always the danger that the material may be aspirated into the lungs. Cobbett showed that organisms could reach the lungs from the air, since cultures taken from the lungs post mortem showed all the organisms usually found in the air. Cultures from other organs remained sterile.

G. Leuenberger[4] contributes records of two interesting cases of placental and of congenital tuberculosis. In the first instance the mother died of tuberculous meningitis and miliary tuberculosis. Tubercle bacilli were found in the fetal liver and numerous miliary tubercles in the placenta. Injection of a small piece of the liver and of the heart's blood of the fetus into a guinea-pig gave rise to pulmonary tuberculosis.

In the second instance the mother suffered from a pulmonary tuberculosis. She aborted. Neither the fetus nor placenta showed any tuberculous changes — but tubercle bacilli were found in the intervillous spaces of the placenta.

Guinea-pigs injected with blood from the umbilical vein developed the disease.

From the study of these two cases Leuenberger draws the following conclusions: When the mother suffers from acute miliary tuberculosis, there can develop numerous miliary tubercles in the placenta and from these tubercle bacilli can penetrate the fetal circulation, and it is also true that without tuberculous changes in the placenta or membranes the bacilli can pass from mother to child; that is, during birth there can be sufficient injury to the chorion vessels to allow the bacilli to pass from the intervillous spaces into the fetal circulation. In view of this fact, immediate tying of the cord of the new-born children of tuberculous mothers is advisable.

K. Peisich[6] doubts the possibility of germinative infection. He says placental disease with penetration of the bacilli into the fetus is the usual method of prenatal infection. He also discredits a specific predisposition to tuberculosis, but regards as more probable a congenital feeble resistance which allows acquired infection. The mode of infection in children varies with age. Young babies are usually infected by their attendants,

either by inhalation or ingestion of the bacilli. The result is a severe pulmonary tuberculosis with rapid generalization. In children up to the school age ingestion of a small number of the bacilli is more usual and the consequence is a benign local form of the disease. Older children are infected as are adults, usually by inhalation of the bacilli.

A. Moeller[7] is of the opinion that an individual can become tuberculous from tubercle bacilli lodged in carious teeth and in the ulcers of unclean mouths.

In order to ascertain the frequency in which tubercle bacilli are found in these locations, he examined two sets of children. The first comprised 194 children with diseased lungs; 153 had carious teeth, 182 had ulcerations in the mouth. Tubercle bacilli were found fourteen times in the carious teeth and the pseudotubercle bacilli twenty-three times; in the ulcerated patches tubercle bacilli were found thirty-five times and the pseudotubercle bacilli forty-two times.

The second series comprised fifty-three healthy children. Twenty-six had carious teeth and forty-one foul conditions of the mouth. No tubercle bacilli were found in the teeth, but pseudotubercle bacilli occurred nine times. In the ulcers tubercle bacilli were found six times and pseudobacilli eighteen times.

While admitting that tubercle bacilli gain access more frequently through the tonsil, the writer still lays stress on the importance of these modes of infection.

O. Ehrhardt[8] reports tuberculosis of the mucous membrane of the mouth and the lower jaw following the extraction of a tooth in a 9-year-old girl. The extraction wound did not heal and there developed in the mucous membrane on either side of the alveolar process a row of grayish-yellow nodules (tubercles). The alveolar process was twice its normal thickness and the cervical glands on the corresponding side were enlarged. The author thinks the movement of the alveolar process indicates an infection at the time of extraction, probably through the instruments. He examined twenty cases of carious teeth in children with tuberculosis and only once found an acid-fast bacillus. This was somewhat thicker than the tubercle bacillus, and may have been Petris' milk-bacillus.

F. S. Mathews[9] says that the tonsil is the probable source of infection of cervical lymph-node tuberculosis, though it may come from a tooth, the pharynx or nasopharyngeal adenoids. No tubercle bacilli were found in fifty-seven tonsils removed for various causes. This confirms the views of Hodenpyl, Wright and Judd, who claim that isolated tuberculosis of the tonsil is rare. Tubercle bacilli were found, however, in five tonsils removed from children suffering from tuberculous cervical adenitis.

C. C. Warden,[11] who found tubercle bacilli in five of twelve tonsils removed according to the usual routine, draws the following conclusions:

(1) Tonsils may oftener be the avenue of tuberculous infection in children than was formerly supposed. (2) The observations of Dieulafoy and Latham are confirmed by this study, namely, that hypertrophied tonsils contain tubercle bacilli in the proportion of 1 to 6. (3) It is possible that tubercle bacilli may assume a vegetative or saprophytic existence in the tonsil.

BACTERIOLOGY

12. Report of English Royal Commissions of Tuberculosis. Tuberculosis, 1911, x, No. 9.

13. Park, W. H., and Krumwiede, C.: Relative Importance of the Bovine and Human Types of Tubercle Bacilli in the Different Forms of Human Tuberculosis. Jour. Med. Research, 1910, p. 205.

14. Litterer, W.: Study of Tubercle Bacilli, Cultivated from Nine Consecutive Cases of Primary Tuberculous Cervical Adenitis. Lancet-Clinic, 1911, cx, 443.

15. Moore, V. A.: Elimination of Tubercle Bacilli from Infected Cattle. Jour. Med. Research, 1911, xxiv, 517.

16. Rothe, F. W.: Tubercular Infection in Children. Deutsch. med. Wchnschr., 1911, No. 8.

17. Abramowski, H.: Contribution to Study of Scrofula. Ztschr. f. Tuberk., 1911, xvii, 488.

18. Eber, A.: Transformation of Human to Bovine Type of Tubercle Bacillus. Zentralbl. f. Bakt., 1911, lix, 193.

19. Howard, W. R.: Relative Importance of the Infection and Transmission of Bovine and Human Tuberculosis to the Public Health. Texas State Jour. Med., 1910-11, vi, 215.

20. Kossel, H.: Relation of Bovine Tuberculosis to Pulmonary Tuberculosis of Man. Deutsch. Med. Wchnschr., 1911, xxxvii, No. 43, 1972.

21. Jessem, F., and Rabinowitch, L.: Presence of Tubercle Bacilli in the Circulating Blood. Deutsch. med. Wchnschr., 1910, xxxvi, No. 24, 1116.

22. Kurashige, T.: Presence of Tubercle Bacilli in the Blood of Tubercular Patients. Ztschr. f. Tuberk. u. Heilstättenw., 1911, xvi, 347.

23. Krause, A.: Bacillemia in Patients not suffering from Miliary Tuberculosis. Ztschr. f. Tuberk. u. Heilstättenw., 1911, xvii, 436.

The following[12] is a summary of the work done by the English Royal Commission of Tuberculosis on the relation between the form of the disease in man and that in animals.

Three groups of tubercle bacilli can be isolated from tuberculous lesions in man. Group I has all the characteristics of the bovine type. Group II of the human type, and Group III has some characteristics of both the others. Human and bovine tubercle bacilli cannot be differentiated morphologically, but they differ in their cultural characteristics and in their power of producing disease in different animals. They can be regarded as different types of the same organism.

Pulmonary tuberculosis is but rarely caused by the bovine type. On the other hand, 50 per cent. of primary lethal abdominal tuberculosis in children is due to the bovine type, and a large per cent. of cervical gland tuberculosis in children is caused by this type also. There can be no doubt that primary abdominal and cervical gland tuberculosis of bovine origin are due to deglutition of tuberculous material and in all probability

this material is milk containing tubercle bacilli. Preventing the ingestion of living tubercle in the milk will, according to the Commission, diminish abdominal and cervical gland tuberculosis among children. The government control of milk and meat must be more widespread in order to prevent the sale of foodstuffs which come from tuberculous animals.

W. H. Park and C. Krumwiede[13] have made a study of human and bovine types of tubercle bacilli in the different forms of human tuberculosis. According to them, tubercle bacilli, isolated from man, fall into two groups, designated as the human and bovine types. These types differ somewhat, especially culturally and in virulence. The groups differ probably because of their residence in different hosts for long periods of time. The evidence of a rapid change of type is incomplete and inconclusive.

The writers collected from the literature records of tuberculosis in which the type of bacillus was identified and compared the findings with those in a group of their own cases. From a study of both these groups they conclude that the incidence of bovine infection is a negligible factor in adults. In children, however, it causes a considerable percentage of cervical adenitis. Further, in young children an appreciable amount of fatal tuberculosis is due to this infection.

William Litterer[14] examined nine consecutive cases of primary tuberculous adenitis to ascertain the type of infecting organism. In five he obtained cultures of the human; in four, of the bovine type. Most valuable for differentiating these types are the reaction curve test in glycerin bouillon and the test of the virulence of the organism to rabbits. (The bovine type is virulent for rabbits, the human is not.) The reaction curves in glycerin bouillon depends on the chemical reaction of this medium, when the bacilli are grown on it. The first change in either instance is the diminution of the amount of acid. In the case of the bovine bacillus, the reaction may become alkaline; in the case of the human type, the neutral point is closely approached and seldom passed.

V. A. Moore[15] has made an extensive study to ascertain the frequency with which tubercle bacilli are eliminated from infected cattle. He found that the milk from cows with tubercular udders contained tubercle bacilli in large numbers, as well as the milk from animals with open pulmonary or intestinal lesions, or lesions of the organs of reproduction, when it becomes contaminated by feces or uterine discharges.

As a rule, the bacilli are not found in the milk from animals in which a careful physical examination discloses no evidence of the disease, although they may give a positive tubercular reaction.

The writer believes that the number of tubercle bacilli in market milk could be greatly reduced and probably eliminated by frequent and thor-

ough physical examinations of dairy cows and the removal of all which show evidence of the disease.

F. W. Rothe[16] performed a number of inoculation experiments on guinea-pigs with pieces of mesenteric and bronchial glands. He wished to ascertain the frequency of bronchial and mesenteric gland tuberculosis in children in Berlin; the mode of infection, and the type of the infecting organism. Glands from 100 children were examined, and twenty-one of the inoculated guinea-pigs developed tuberculosis. From twenty of these the human bacillus was isolated and from one the bovine. From this study Rothe concludes that 20 per cent. of the children in Berlin are infected with tuberculosis; that the preponderance of infection is through the respiratory tract, and that the bovine type of bacillus plays a part secondary to the human type.

A. Ebers[18] believes that in a number of instances he succeeded, by the passage through rabbits and guinea-pigs, in transforming tubercle bacilli obtained from a human source, and having the characteristics of the human type, into bacilli with all the characteristics of the bovine type.

W. R. Howard[19] enumerates the following ways in which human beings can become infected with the bovine tubercle bacillus: (1) By the use of milk from tuberculous cows and its products. (2) By the use of infected meats, improperly cooked or carelessly handled. (3) Through drinking water from wells where tuberculous animals water. (4) Through flies in the immediate proximity of infected herds carrying the infection to unprotected foods.

H. Kossel[20] examined 709 phthisical patients and found the bovine type of bacillus alone probably three times — surely twice; the mixed form once; and the human type 705 times.

These numbers speak for the fact that phthisis is spread from man to man.

F. Jessem and L. Rabinowitch[21] have undertaken a series of researches to verify the findings of various writers that tubercle bacilli occur in the blood not only in the advanced, but also in the early stage of tuberculosis, and they have been able to detect bacilli in the blood, even in patients afflicted in a mild degree. They believe blood examination a very important diagnostic measure, especially when the sputum cannot be obtained.

T. Kurashige[22] was able to demonstrate tubercle bacilli in 100 per cent. of 155 mild and severe cases of pulmonary tuberculosis. The penetration of the bacilli into the blood does not always give rise to miliary tuberculosis. The finding is of diagnostic but not of prognostic value.

A. Krause[23] has not been able to demonstrate tubercle bacilli in the blood as frequently as other observers. He does not think the prognosis of these bacilli carriers absolutely bad.

DIAGNOSIS

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26. Pollack, R.: Tuberculosis in Nurslings. Beitr. z. Klin. d. Tuberk., 1911, xix, No. 2.

27. Klose, E.: Cutaneous Bovine Tuberculin Reaction. Deutsch. Med. Wchnschr., 1910, No. 48, p. 2239.

28. Wallenstein, P. S.: Diagnostic Importance of the Cutaneous Tuberculin Reaction. Berlin klin. Wchnschr., 1911, xlviii, No. 10.

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E. Feer[24] studied the v. Pirquet and conjunctival tuberculin tests in children. He vaccinated 2,000; of these, 171 were tuberculous, ninety-seven suspicious and 1,732 free from tuberculosis; 163 of the tuberculous children, sixty-two of those suspicious, and 277 of those free from tuberculosis gave positive reactions.

A positive v. Pirquet reaction is without doubt specific for tuberculosis. Especially valuable is the test during the first three years; after this time a negative result is of more value than a positive one. Care must be taken to distinguish the spurious from the true reactions. Reactions which disappear in twenty-four hours are not specific.

As to the conjunctival reaction, Feer believes it more often indicates an active tuberculosis than the v. Pirquet, but he does not recommend its use, as severe conjunctivitis and phlyctenules often develop.

L. Hamman and S. Wolman[25] have made the conjunctival and v. Pirquet tests in 1,500 patients. They sum up their opinion of the relative importance of the two tests as follows: "As the specificity of the reactions is now well established in the sense that only patients who have at some time been infected by tubercle bacilli can react, the test shows that a large number of healthy people have been so infected, but as the test remains positive, even when the infection has been overcome, the story it tells is of absorbing interest to the clinician only when the patient is still in his infancy. Reactions during the first year or two of life mean that the patient has been only recently attacked and must be strengthened for combat with the invader, but if the test is negative and the child not moribund or suffering from measles, any suspicions of tuberculosis are dispelled.

"The conjunctival test, if only safe solutions are used, is not nearly so sensitive as the cutaneous test. In event of a negative reaction entire reliance must be placed on other methods of investigation."

R. Pollak[26] agrees that a negative cutaneous reaction in nurslings speaks with the greatest possibility against the existence of a tuberculous focus, which, however, cannot absolutely be excluded. If the reaction is negative, the more delicate "stick-reaction" must be employed. According to Hamburger, this reaction is obtained by injecting beneath the skin 0.1 to 1 mg. of tuberculin. It must not be injected later than the second or third day after vaccination or the increased sensitiveness to tuberculin caused by the vaccination may lead to a general reaction. A positive v. Pirquet reaction proves not only the presence of a tuberculous focus, but that the process is an active one, because latent tuberculosis does not exist in nurslings.

E. Klose[27] vaccinated 120 children, suspected of having tuberculosis, with human and bovine tuberculin. Of these, forty did not react to either; the others reacted similarly to both. Klose offers two explanations. Either the antigens of human and bovine tuberculin are identical or there are two kinds of antibodies formed in the body and each reacts with its particular antigen.

The writer believes that from the results obtained by vaccinating with both tuberculins one can decide which should be used as a therapeutic measure.

P. S. Wallenstein[28] believes the more latent the tuberculosis, the stronger must be the concentration of the tuberculin to cause a reaction and that this usually runs a course of a "late reaction." The more the process leans toward an initial active stage, the weaker should the concentration of the tuberculin be; even a one-eighth per cent. solution can cause a reaction. The more manifest the tuberculosis, all the more

concentrated must be the tuberculin, and in very advanced cases the reaction is negative even with 100 per cent. solutions.

St. Engel[29] declares that the intracutaneous tuberculin test has numerous advantages over other tuberculin tests. He uses one drop of a 1/1,000 solution of old tuberculin and if no reaction follows, increases the strength to 1 per cent., 10 per cent., and 100 per cent. In some instances the patients will react to the larger dose and not to the smaller. The subcutaneous test fails with febrile patients and the cutaneous with patients whose susceptibility to tuberculin is reduced, but the intracutaneous method succeeds when these others fail.

G. Rovere[34] repeated the conjunctival test in thirty non-tuberculous children under 2 years of age, to ascertain whether a local "hypersensitiveness" could be obtained. Each child was tested eight to ten times. The results were negative and substantiated the findings of other authors, that local "hypersensitiveness" cannot be produced by repeated instillations of tuberculin into the eye.

Barheim-Karrer,[35] discussing the condition of hypersensitiveness of the skin to the v. Pirquet test, asserts that children suffering from eczema can give a positive reaction, when there is no focus of tuberculosis. A differential diagnosis can be made only by a second vaccination which is negative in eczema and markedly positive in tuberculosis. Cases of hypersensitiveness to the v. Pirquet test, as described by this writer, fall into two groups, the first including those with severe local symptoms; the second, those with general symptoms.

In the first group the papule is especially large, intensely red, with sometimes a vesicle in the center, rarely an ulcer. The lesions last much longer than in ordinary reactions.

In the second group, beside the severe local manifestations, lesions have occurred in other parts of the body, and phlyctenules, conjunctivites and rhinitis have all been observed. The general eruptions are especially interesting and resemble measles or scarlet fever, as well as urticaria and erythema urticatum. Their incubation stage is not regular; they may appear ten hours or ten days after the vaccination.

J. Sorgo[36] has added a contribution to the question whether the tuberculous infected organism possesses a specific sensitiveness to tuberculin only or to other toxins as well. He vaccinated the same individual with tuberculin, diphtheria, and dysentery toxin. The reaction was marked in all three instances. Sorgo comes to the conclusion that the v. Pirquet test depends on a local sensitiveness of the skin to the toxin. This sensitiveness is increased in the majority of non-cachectic infected individuals for tuberculin and other toxins.

E. Tezner[37] ascertained that all children who gave positive v. Pirquet reactions reacted also with colitoxin. He absolutely denies the specificity

of the v. Pirquet reaction and thinks it depends on the reaction susceptibility to the protein of any or all bacteria with which the organism is infected.

F. Rolly[38] performed a number of experiments to see if irritation of the skin would interfere with the v. Pirquet tuberculin reaction. Also, if other toxins as those of cholera, dysentery, and pyocyaneus would give reactions. He irritated the skin two hours before vaccination with mustard plasters, iodin, naphthol and cantharides, and found that the reactions to tuberculin were then much less marked and often negative.

Of the children vaccinated with the various toxins, 16 per cent. gave positive results. These could not possibly be anaphylactic or specific reactions as the children had never suffered from these diseases. He found also that their cutaneous reactions not only for tubercle, but for the other toxins, became negative during the course of various diseases.

Rolly believes that cutaneous susceptibility follows other laws than that which governs general susceptibility.

Hanna Hirschfeld[39] examined the blood in the various forms of tuberculosis in children to determine the relation of the white blood cells. She found that prognostic favorable cases show a tendency to lymphocytosis, at times to eosinophilia. Unfavorable cases always show absence of eosinophil cells. Tuberculin injections do not modify the picture.

J. L. Leopold and T. Rosenstern[40] believe the skin lesions are an aid in diagnosing tuberculosis in infancy. They especially call attention to the papulonecrotic tuberculids, which consist of slightly raised papules varying in size from a pinhead to a millet seed. At first they are red but later become brownish and show a scale or crust in the center. The lesions may appear on any part of the skin, but the sites of election are the arms, the lower back and the extensor surface of the lower extremities. The authors found them in 40 per cent. of their cases.

T. C. Noeggerath and v. Salle[41] have studied "Head's zones" in early tuberculous lesions of children. Head found that, when a segment of the spinal cord is irritated through disease of any internal organ, irritation is transmitted to the skin area supplied by nerves from that spinal segment. The technic for eliciting Head's zones is simple. The skin is stroked gently with the blunt end of a pin held vertically. The patients tell when there is pain and when not. Now and then the pain is so severe that the muscles twitch.

The writers studied this sign in forty-six children, of whom twenty-four were clinically suspected of tuberculosis. Of these twenty-four, sixteen showed Head's zones of sensibility; in eight they were absent. In twenty children with no lung-findings the sign was absent.

The areas found sensitive were those supplied by nerves from the second, third and fourth dorsal segments and fourth cervical areas, which,

according to Head, suggest disease at the base of the lungs, and of the bronchial glands.

The writers think from their examination that Head's zones in early or incipient tuberculosis have the same significance as in other diseases.

E. Mayerhofer and R. Neubauer[42] think testing the spinal fluid for organic material by the potassium permanganate method is a very important procedure in the diagnosis of tuberculous meningitis. The method was explained in a former contribution of Mayerhofer, as follows: One cubic centimeter of spinal fluid is mixed with 50 c.c. of distilled water; 10 c.c. of a 25 per cent. sulphuric acid is added, and when these are brought to a boil, 10 c.c. of one-tenth normal solution of potassium permanganate. The whole is kept simmering for ten minutes and then 10 c.c. of one-tenth normal solution of oxalic acid is added, which decolorizes the fluid. Then more of the permanganate solution is added drop by drop, until the first drop leaves a distinct rose color. The number of cubic centimeters of the permanganate solution indicates the amount of organic material.

The spinal fluid from 100 cases, mostly of tuberculous meningitis, was tested. A few cases of meningitis of other origin, a few of convulsions in children, and two cases of poliomyelitis were also tested. In all cases of tuberculous meningitis the index for organic matter was very high, from 2 to 30 and even 37.3. Exudates of tuberculous pleurisy and peritonitis gave the same high figures.

From their studies the authors conclude that a tuberculous meningitis cannot exist without a high reducing power of the spinal fluid to potassium permanganate, although not all cases with high reducing power are those of tuberculous meningitis. The reducing power occurs early in the course of the disease and so is a valuable diagnostic aid.

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K. E. Ranke[43] differentiates three forms of pulmonary tuberculosis in children: (1) Phthisis, which is analogous to that in the adult, is relatively rare, and has a more favorable diagnosis. (2) Generalized tuberculosis, which occurs most frequently in the first and second years and has an absolutely bad prognosis. (3) The so-called hilus catarrh. The children affected with this form are anemic, have scrofulous symptoms, give positive tuberculin reaction, and frequently suffer from tracheal and bronchial catarrh. Examination of the lungs shows changing, localized râles in the apices, with eventually exaggerated, weakened or rough breathing, and lessened resonance on percussion.

R. Pollak[45] has made a study of the clinical manifestations of tuberculosis in nurslings, and thinks the primary lesion at this age is a bronchial gland tuberculosis. This primary defect is not usually diagnosed because of its mild character. Pollak mentions, however, that lately Sluka has reported that it can be regularly discovered by means of the Roentgen rays. Clinical symptoms, says Pollak, manifest themselves only when the glands have reached a certain size, and the two cardinal ones are the high-pitched cough, like the bark of a young dog, and an expiratory crow. Next in importance for diagnosing tuberculosis in nurslings are the skin lesions. Besides lichen scrofulosorum, scrofuloderma and erythema induratum, the writer has frequently found the small papulosquamous tuberculid.

Tuberculous meningitis occurs early in the course of tuberculosis in nurslings. The author found that instances of this rarely gave a history pointing to a primary bronchial gland tuberculosis; that it was discovered post mortem but gave no clinical signs.

Pollak also mentions a latent form of tuberculosis in nurslings. He says that tuberculosis should be suspected in infants properly fed, who, without symptoms on the part of the digestive system, have a stationary weight-curve, or one which runs downward. The only evidence in these children is the positive cutaneous test and the presence of skin tuberculids.

Tuberculosis in nurslings, according to this author, has not so bad

a prognosis as was formerly supposed. The patients frequently survive the first year. The prognosis depends on the source of infection. Infants infected from a virulent source usually develop a severe form of the disease; those infected from a chronic phthisis, or mild form, suffer from a milder curable type. Children who are infected during the first year and survive, develop a typical habitus. They are pale and thin, with flabby muscles. They have long eye-lashes and hair on the temples and between the shoulder blades. The writer has watched this habitus develop in children infected during the first year of their life.

B. Huguenin[47] is of the opinion that the bronchial glands can become infected without any lesion of the lungs. This can take place in two ways, either from a descending or ascending gland tuberculosis or by taking up the bacilli from the intact mucous membrane of the respiratory tract. In the mildest cases the enlarged glands cannot be detected by physical examination. The disease runs the course of a subfebrile or febrile bronchitis, with anemia and emaciation. There are no lung findings and no bacilli in the sputum. The lesion in the glands either becomes encapsulated, caseated or calcified; that is, there is a relative cure, or it progresses and ruptures into a vein with a resulting miliary tuberculosis. The author also describes a hilus tuberculosis resulting from the rupture of the gland into a small bronchus. If the gland becomes secondarily infected with any of the pus organisms, it suppurates and may rupture into the esophagus, pericardium or pleura.

H. French[48] believes that enlarged tuberculous lymph-nodes situated at the bifurcation of the trachea are a cause of chronic cough among children. The right bronchial node is the one most frequently affected and any enlargement of this directly irritates the phrenic nerve and causes paroxysms of coughing. In a number of cases it is possible to demonstrate the enlargement by means of the Roentgen rays. It is important to recognize this as a cause of chronic coughs, so that the general hygienic measures to correct tuberculous infections may be instituted.

H. Stoll[49] thinks it surprising that the diagnosis of bronchial node tuberculosis has not received more attention. To ascertain the reliability of a number of signs regarded as indicative of this condition, he has studied 175 children and compared the results of his examination with the skin tuberculin test.

On inspection the most important sign to be seen is the dilated veins over the anterior aspect of the thorax. On palpation tenderness over the manubrium and sternocostal junction is suggestive, but these tender areas appear less frequently than tenderness over the spines of the upper thoracic vertebræ. Dulness obtained by percussion over the upper part of the sternum has little diagnostic value but interscapular or vertebral

dulness is of decided value, as well as vertebral bronchophony. This is elicited by placing the bell of the stethoscope directly over the vertebræ and having the patient whisper slowly the words, "three-thirty-three." Normally, the tracheal character of the respiratory murmur ceases at the seventh cervical vertebra, but, when enlarged bronchial nodes are present, it is continued downward over the thoracic spines.

The *x*-rays are very valuable in diagnosis. The writer has been able to compare the physical findings with the *x*-ray plate in fifty instances. Calcified nodes are recognized by their sharp, clear shadow, while cheesy ones give a shadow of less density and less clearly marked boundaries. The earliest evidence of the bronchial nodes seems to be a shadow which extends from the anterior tip of the third rib on the right side upward to the first rib. This shadow extends to the right of the vertebræ for a variable distance. A chain of nodes can often be seen along the right border of the heart. The ones about the trachea give a more diffuse shadow than those at the hilus. Quite commonly an enlarged node is observed close to a bronchus.

C. Leiner and F. Spieler[50] describe the various forms of disseminated tuberculosis of the skin in children.

Lupus Vulgaris Disseminatus.—The eruption of this form is characterized by nodules from the size of a pinhead to that of a pea, bright red, bluish or brownish in color, and sharply defined from the surrounding tissue. They are of soft consistency and in the early stage of the disease the skin is drawn taut over the upper surface. The eruption is explosive and over night the entire body can be covered with small nodules or they may appear in crops. The lesions may remain unchanged for months or may at any time disappear, leaving behind slightly pigmented superficial scars. Or, by the apposition of new nodules large plaques may form. They do not, however, lead to destruction of the underlying tissues.

Gommes Scrofuleuses Disséminées.—The primary efflorescences are nodules which develop in the subcutaneous or cutaneous tissues, without any inflammatory reaction. They are hard at first, then soften, and break through the thin bluish-red overlying skin. It takes weeks or months for the thin fluid with cheesy masses to discharge; then scarification sets in, with the formation of bluish-red or bluish scars.

This form frequently appears at the end of the first year of life, and is the first manifestation of a latent tuberculosis.

Lichen Scrofulosorum.—The lesions consist of pinhead to needle-sized papules, located around the hair follicles. They resemble lesion of lichen planus.

The papulonecrotic tuberculid is the best-known of all the tuberculids. The primary lesion is a small brownish or bluish nodule which soon becomes grayish white, and indented in the center. A few drops of

creamy pus are exuded and the lesion is covered with a dry crust or scale. As the center dries, the lesion flattens, loses its nodular character finally, loses its crust, and completely heals, leaving behind a characteristic scar, with central depression and a peripheral area of pigmentation.

Erythema Induratum resembles erythema multiforme and must be differentiated from this disease. The large, hard nodes, of livid red or violet fading into the normal skin, are usually situated on the lower extremities. They differ from those of erythema multiforme in not being painful or itching. They usually occur in young girls with a scrofulous diathesis.

Disseminated Miliary Tuberculosis of the Skin.—This eruption scattered over trunk and extremities has a purpuric character. The single lesions are pinhead to lentil sized, livid red or reddish brown, and do not completely disappear on pressure. The center is sometimes depressed and covered with a crust or scale. As the lesions disappear, they leave behind an indented pigmented spot. This form is a manifestation of a general miliary tuberculosis.

The prognosis of the disseminated tuberculids is not necessarily unfavorable. Even the miliary tuberculids may heal before the inevitable death of the patient. Their diagnostic value is great, for they are often the first manifestations of the disease.

M. Lafarcinade[51] describes a "somnolent" form of tuberculous meningitis, which occurs in young infants. Its onset is gradual; for the first four or five days the child shows a tendency to sleep more than usual, and this becomes more noticeable, until he awakens only to nurse and immediately falls asleep again. Ultimately the sleep is continuous and the child becomes comatose; the eyes remain open. Emaciation is progressive from the beginning. If the pulse is examined several times a day, it is found to vary from hour to hour, perhaps 90 one hour, then 140 to 160. There may be diarrhea and vomiting.

After two weeks of these symptoms the picture is as follows: The child lies in his cradle, usually on his back, with no expression on the face, with the eyes half closed; he cannot be roused; the pulse is hard, regular, 100 to 160; the temperature is subnormal, but rises toward the end. The coma deepens and death occurs in eight to ten days.

W. G. Archangelsky[52] reports a case of tuberculous meningitis in an 8-year-old child who recovered. Lumbar puncture on admission gave a clear fluid, containing numerous mononuclear cells and tubercle bacilli. On the twenty-third day the fluid was normal.

The writer has made a very careful study of the literature and has found several reports of recovery from tuberculous meningitis. In some cases the diagnosis was substantiated at autopsy, when, the child having died from other causes, healed tubercles were found. In other cases it was

based on the character of the spinal fluid and the finding of tubercle bacilli.

Archangelsky emphasizes the value of lumbar puncture as a therapeutic agent, since, on the one hand, it diminishes the cerebrospinal pressure, and, on the other, eliminates the tubercle bacilli and their toxins.

He says that the rare occurrence of these cases does not alter the prognosis of the disease as it is known, but is of theoretical interest and spurs the physicians further to work out the pathology and therapy of diseases of the meninges in general and tuberculous meningitis in particular.

J. Cassel[53] has observed forty-five cases of tuberculous peritonitis in children. Of these thirty-one were not operated on, eight recovered, nine died, and the fate of the others is not known. Of those operated, six recovered, six died.

Cassel says that if, after a proper general régime has been carried out for weeks, a febrile stage develops with increased swelling of the abdomen and emaciation, he does not hesitate to turn the patient over to the surgeon. But he does not advise operation if hygienic and dietetic treatment cannot be continued after the operation.

The following interesting case of tuberculous peritonitis is reported by A. Galliot:[54] A rachitic child of 6 years had shown signs of tracheobronchial adenopathy, mucomembranous enteritis, with meteorism, and slight fever; but for eighteen months he had felt well. On the night of January 8 he showed vague symptoms which were attributed to an indigestion. The following day there was vomiting with pains in the abdomen and a temperature of 38 C. On the tenth the pain became localized in the right iliac fossa. There was decided resistance and tenderness over McBurney's point. A diagnosis of appendicitis was made and the abdomen opened. The peritoneal cavity contained a yellowish fluid. The intestinal coils were adherent, as well as the appendix, which was in the retrocecal fossa. On the peritoneum of the cecum were small calcareous masses and typical tubercles. None occurred on the appendix. The appendix was extirpated and the abdomen closed.

J. H. Bradshaw[55] describes a case of tuberculous peritonitis, due probably to direct intestinal infection. The child, an 18-months-old baby, was well-developed and had never been sick. One day he picked up from the floor a walking-stick and sucked the ferule end. Soon afterward the typical picture of a severe peritonitis sicca developed. Laparotomy was performed, the abdomen being simply opened and closed. Improvement was transitory, then the child failed rapidly. A second operation was performed. Adhesions were broken up and after a futile attempt to free the ileocecal region, which was filled with tubercular

masses, the abdomen was closed again. The child recovered, is now 3 years old, and in good health.

Bradshaw believes tuberculous peritonitis in children arises chiefly from an intestinal infection and that milk is a frequent source of it.

P. Vignard and L. Thévenot[56] discuss tuberculosis of the testicles in children. They report three cases. The first patient was 12 years old. There had been a swelling in the scrotum for about a month. Examination showed a testicle of normal size, but a large nodular indurated epididymus. The vas deferens was also indurated. Epididymectomy and resection of the vas resulted in recovery.

The second patient was a 14-months-old baby. Both testicle and epididymus were involved; a fistula from the testicle discharged a yellow serous pus. Although an operation was performed, the child died six months later.

The third patient was aged 11 months; the testicle, epididymus and vas deferens were all involved. The child died of tuberculous meningitis shortly after the operation.

According to these writers, tuberculosis of the testicles may be primary or secondary. The primary disease differs from that which occurs in adults in involving both testicle and epididymus. The secondary form is much more extensive. It is usually bilateral and involves, besides the testicle and epididymus, the prostate, inguinal, iliac, and lumbar glands.

The onset may be sudden and acute and suggests an acute gonorrheal orchitis, or may be slow, and remain unsuspected until revealed by chance or by rupture of an abscess.

M. Cabannes[57] reports the history of a child attacked by a primary tuberculosis of the conjunctiva, the result of direct inoculation. She was kissed on the eye by a patient suffering from phthisis and a month later a vegetative, ulcerative conjunctivitis developed, with suppuration of the submaxillary and postcervical glands. As a control for diagnosis, Cabannes inoculated a fragment of the vegetation into the anterior chamber of the eye of a rabbit. The animal developed a keratitis, which healed spontaneously; but three weeks later a tuberculous lesion developed at the point of inoculation.

W. Milligan[58] asserts that tuberculous otitis occurs more frequently than is usually supposed, and, because of its high mortality and the irreparable damage which it does to the ear, it ought to be recognized early and subjected to suitable treatment. The author thinks that 20 per cent. of the children under 6 years of age suffering in the hospitals from a purulent otitis owe the disease to a tuberculous infection. The infection probably passes through the pharyngeal or nasopharyngeal mucosa.

There are no particular subjunctive symptoms pathognomonic of tuberculous otitis. The clinical picture is usually as follows: There is no pain, and examination of the drum reveals no inflammatory reaction, but frequently two perforations or more.

There is early enlargement of the peri-auricular glands and facial paralysis. The discharge is copious, thin, sanious and extremely fetid. The tuberculin reactions are of value, as aids to diagnosis. The prognosis must be guarded, for the death-rate is as high as 50 per cent.

Operative treatment is necessary. If the infection spreads to the glands, tuberculin treatment is advised — 1/10,000 to 1/20,000 mg. controlled by the opsonic index.

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J. B. Hawes[59] emphasizes the importance of caring for children with tuberculosis and training them in the right way of living. He deplores the fact that there is no place in Massachusetts where children with open tuberculosis can be cared for and that there are only one or two open-air schools where anemic pretuberculous children can get fresh air and sunshine, in addition to a knowledge of reading, writing and arithmetic.

He suggests that one means of forcing the state and local authorities to provide for these children is to make application for their admission to state sanatoriums. The strongest argument which the board of these institutions can use before the legislature is a long list of applications from children for whom there is no place.

He also suggests earnest and intelligent cooperation with the local school board to provide fresh air rooms for pretuberculous children, and that tuberculosis exhibits should be placed in the schools and demon-

strated by doctors, nurses and social workers to inform the children about the disease and its prevention.

A series of articles has lately been published on open-air schools. In these various institutions the equipment, methods of feeding, examination and teaching are more or less similar. J. Perkins describes the school in Providence, R. I. The exposure of the open side of the room is south. The feet of the children are protected by sitting-out bags, such as are commonly used for tuberculous patients. The children wear caps and mittens. They are given some form of nourishment, e. g., hot milk or soup at 10 o'clock. They bring a lunch with them and eat it at 12 o'clock, having it supplemented by hot cocoa, pudding, or a substantial stew. The lunch is served at a regular table, the children being taught to set the table, clear it, and wash the dishes. They must wash their hands and faces and brush their hair before eating and cleanse their teeth after eating. It is important for the success of the work that the children be visited in their homes and the home conditions studied and improved.

In Boston's hospital school for tuberculous children,[68] besides the incipient cases, more advanced ones were taken. The entire staff consisted of three physicians, a nurse, a social worker, a dietitian, five teachers, a cook, and two maids. The children were divided into three groups. Those in the first were required to follow the regular routine school work. The second group was intermediate. The third was given a minimum amount of study and exercise.

This school has been closed, but the open air rooms in the Boston schools are a direct outcome of the work done there.

TREATMENT

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73. Raw, N.: The Treatment of Pulmonary Tuberculosis with Bovine Tuberculin. Lancet, London, 1911, No. 180, 929.

74. Gouraud: Tuberculin Therapy in Childhood. Arch. de méd. des enf., 1911, xv, 744.

75. Jochmann, G., and Moellers, B.: Treatment of Tuberculosis with Albumose-Free Tuberculin, Deutsch. med. Wchnschr., 1911, xxxvii, No. 28, 1298.

76. Comby, J.: Recalcification Treatment of Tuberculosis in Children. Arch. des méd. des enf., 1911, xix, 213.

77. Sobotta, E.: Tubercular Predisposition and its Conquest. Ztschr. f. Tuberk. u. Heilstättenw., 1911, xvii, 230.

78. Bernheim, S., and Dieupart, L.: Treatment of Tuberculosis with Radio-active-Iodin Menthol. Ztschr. f. Tuberk. u. Heilstättenw., 1911, vii, 440.

79. Goodall, H. S.: Results of Sanitarium Treatment in Children. Boston Med. and Surg. Jour., 1910, p. 432.

80. Bardenheuer, B.: Sunlight in the Treatment of Surgical Tuberculosis. Deutsch. Ztschr. f. Chir., 1911, cxii, No. 1-3.

81. Joachimsthal, G.: What Paris is doing for Children with Bone and Joint Tuberculosis. Berlin. klin. Wchnschr., 1911, xlviii, No. 8.

T. Escherich[71] believes that the fight between the tuberculous infection and the organism is dependent on the formation of anaphylactic antibodies and on the general resistance of the patient. Escherich suggests the administration of tuberculin, not in large doses, as Schlossman and Engel advise, but in doses just large enough to stimulate the production of anaphylactic bodies. The dose is 0.001 to 0.002 c.mm. injected once or twice a week for two or three months. As this dose is seldom followed by a rise in temperature or untoward symptoms, it can be advantageously used in an ambulatory clinic. After the first or second injection the general condition is usually improved. After being treated two months the children should be sent to the country. Escherich has given this method a thorough trial and has met with satisfactory results. He does not use it with patients in advanced stages, but in those who are brought with indefinite signs of the disease, as pallor, loss of appetite, general debility, loss of weight, and a positive v. Pirquet reaction.

In conclusion, he says, if tuberculin is to have a permanent place in the treatment of tuberculosis of childhood, it must not be used, as it formerly was, as a last resort, but in latent or incipient cases, and should be given in carefully regulated doses.

According to L. C. Ager,[72] the curative effects of tuberculin are due to the stimulation of the body cells and not to any direct action on the tuberculous focus. The type of cases in which tuberculin is most useful are the chronic ones which do not yield to ordinary treatment. Under hygienic treatment patients of this class improve, but sooner or later suffer a return of symptoms with increased physical findings.

The organism needs stimulation, which is effected by the use of tuberculin. It is best given in small doses, 1/5,000 or 1/1,000 mg. daily, for a period of ten days, or two weeks. The daily dose is increased, but must not exceed 0.01 mg. After such a course of treatment there is usually a definite and progressive improvement for several months, then an exacerbation, but much slighter than before the tuberculin treatment. This is an indication for another period of tuberculin treatment. Three or four such courses ordinarily bring about a cure. Patients should be in a sanatorium or hospital and also receive the usual outdoor and hygienic treatment.

N. Raw[73] advises the use of bovine tuberculin in the treatment of tuberculosis. He says it is less irritating and less likely to produce reactions when given in large doses.

Gouraud[74] believes that tuberculin is of great service in treating tuberculosis of children, especially in scrofula, bone and glandular tuberculosis, and tuberculous cachexia, but it is useless in pulmonary tuberculosis. Initial doses are 0.001 to 0.01 mg., which can be rapidly increased to a gram.

G. Jochmann and B. Moellers[75] use a tuberculin which is obtained by growing the tubercle bacillus on a medium deprived of albumose. The authors maintain that this tuberculin is less toxic than the old tuberculin, causes little or no reaction, and can be increased rapidly in dosage.

J. Comby[76] discusses Ferrier and Sergent's recalcification method of treating tuberculosis in children. This seeks to combat the calcium excretion and to introduce into the organism salts of calcium capable of being fixed in the tissues. In the first place, the dyspepsia so frequent in overfed tuberculous patients is corrected by lengthening the interval between meals and regulating the diet. Foods usually given dyspeptic children are allowed, as milk dishes, eggs, boiled fish, grilled or roasted meats, purees of vegetables, dried fruits, toasted or dry bread. Fats, alcoholic drinks, foods easily fermentable, like cheese, are forbidden. Calcium-containing mineral waters are given freely. The following tablet is given between meals:

	Gm.
Calcium carbonate	0.30
Tricalcium phosphate	0.50
Sodium chlorid	0.15
Calcined magnesia	10.00

Cod-liver oil, acid medicaments and syrups are rigorously forbidden. Alternating with the tablets, arsenate of soda is prescribed as follows: The child takes twenty tablets in ten days; then for six days a teaspoonful of the following: arsenate of soda, 0.03 gm., aquæ, 100 c.c. Then the tablets again.

Comby speaks highly of this treatment, although he emphasizes the fact that hygienic treatment must not be forgotten, but must be placed first.

According to E. Sobotta,[77] the assertions of the French writers regarding demineralization in tuberculosis has been confirmed by other writers. Phosphaturia and softening and caries of the teeth are evidence of this demineralization. Sobotta calls attention to the fact that tuberculosis is rare in regions where the water has a high calcium salt content. He believes that lime should not only be given freely to tuberculous patients, but that a predisposition to the disease should be fought by the administration of lime salts.

S. Bernheim and L. Dieupart[78] treated seventy-five patients with tuberculosis with Szendeffy's radio-active iodin menthol solution. The preparation consists of the following: peptonized iodin, 0.75; menthol, 0.06; radium barium chlorid, one-tenth of a drop in ether solution. Each day 1 c.c. of the solution was injected deep into the muscles for thirty days, then repeated two or three times after an interval of fourteen days.

No intolerance of the drugs was shown even in children. The writers are very enthusiastic about their results. Gain in weight was constant, the appetite increased, and the expectoration diminished.

H. S. Goodall[79] summarizes the results in the treatment of tuberculous children at Stoney Wold Sanitarium during the first six years of its existence. In all, 120 children were discharged. The results were better than those obtained among adults under the same conditions. All who were discharged as apparently cured or with the process arrested, at present are as well as when discharged, or better. The author found that little girls between 6 and 12 years yielded more readily to treatment than any other group. Boys between 13 and 16 show a percentage of good results above the girls of the same age and come next to the little girls.

B. Bardenheuer[80] tried the effect of systematic sunlight in the treatment of surgical tuberculosis and his experience has convinced him that far better ultimate results can be obtained with sunlight without mutilating operations. Bardenheuer had the lesions exposed to sunlight for fifteen minutes three times a day, rapidly increasing to two or three hours morning and afternoon, or all day long except in midwinter. As he had no facilities for sun baths, he simply had the patients placed at the open window, where the sun could reach the lesion.

PATHOLOGY

Enteroptosis in Children

(R. R. Smith, Journal A. M. A., February 10, 1912, p. 385.)

After reference to a former paper in *The Journal* (Nov. 26, 1910, p. 1860), and redescribing the symptomatology of the enteroptotic woman of the true or non-acquired type, Smith says that careful inquiry as to the history of the well-developed cases will trace it back to early childhood, i. e., they will tell you they were thin, pale and perhaps more or less nervous even at that early period. He has since studied 109 female children ranging in age from birth to 13 years. The height was obtained and, following the routine used with adults, the size of the middle zone of the trunk was estimated by obtaining the index of Becher and Lenhoff, which gives the relative capacity of the upper abdomen in a general way. This is obtained by dividing the jugulopubic distance by the waist circumference and multiplying the result by 100. After this the general state of nutrition is observed, the attitude of the child, the size and shape of the thorax and the epigastric angle. The kidneys were palpated where possible and the position of the lower border of the stomach determined by the gas method and percussion. Some examinations were necessarily incomplete. A number of photographs were obtained and in twenty-four cases the *x*-ray was used. He was also enabled, through the kindness of Dr. R. W. Lovett, to examine a number of children in the various Boston institutions, but his work was done largely in the D. A. Blodgett Home for Children in Grand Rapids. From his study he concludes that the enteroptotic

habit of adult life is seen in its fundamental characteristic in the frail child—frailness, lack of fat, slenderness of muscle and lack of vigor in bodily development. The actual collapse of the thorax and the lessened capacity of the upper abdomen is observable only in the older children, and then only exceptionally, but the muscular insufficiencies of later life are common in frail children. The prolapse of the kidneys, stomach, colon and the intestines, which is seen in the adult, is not pronounced in children under 12 except in rare instances and then only as a late development. He thinks that the consequences of this condition in childhood are chiefly seen in the adult and much can be done to prevent them. If this can be uniformly done there will be many less weak and physically incompetent women.

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